TYRA

Corporate Deck

May 2024

Disclaimers

Forward-looking statements and market data

We caution you that this presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding our future results of operations and financial position, business strategy, research and development plans, the anticipated timing and phase of development, costs, design and conduct of our ongoing and planned preclinical studies and clinical trials for our product candidates, the timing and likelihood of regulatory filings and approvals for our product candidates and the potential benefits of regulatory designations, the potential to develop product candidates and the potential safety and therapeutic benefits of our product candidates, our ability to commercialize our product candidates, if approved, the pricing and reimbursement of our product candidates, if approved, the timing and likelihood of success, plans and objectives of management for future operations, and future results of anticipated product development efforts, are forward-looking statements. In some cases, you can identify forward-looking statements by terms such as “may,” “will,” “should,” “expect,” “plan,” “anticipate,” “could,” “intend,” “target,” “project,” “contemplates,” “believes,” “estimates,” “predicts,” “potential” or “continue” or the negative of these terms or other similar expressions. The inclusion of forward-looking statements should not be regarded as a representation by us that any of our plans will be achieved. Actual results may differ from those set forth in this presentation due to the risks and uncertainties inherent in our business, including, without limitation: we are early in our development efforts, have only recently begun testing TYRA-300 and TYRA-200 for oncology in clinical trials and the approach we are taking to discover and develop drugs based on our SNÅP platform is novel and unproven and it may never lead to product candidates that are successful in clinical development or approved products of commercial value; potential delays in the commencement, enrollment, and completion of preclinical studies and clinical trials; results from preclinical studies or early clinical trials not necessarily being predictive of future results; our dependence on third parties in connection with manufacturing, research and preclinical testing; we may expend our limited resources to pursue a particular product candidate and/or indication and fail to capitalize on product candidates or indications with greater development or commercial potential; acceptance by the FDA of INDs or of similar regulatory submissions by comparable foreign regulatory authorities for the conduct of clinical trials of TYRA-300 in pediatric achondroplasia; an accelerated development or approval pathway may not be available for TYRA-300 or other product candidates and any such pathway may not lead to a faster development process; later developments with the FDA may be inconsistent with the minutes from our prior meetings, including with respect to the design of our planned Phase 2 study of TYRA-300 in ACH; unexpected adverse side effects or inadequate efficacy of our product candidates that may limit their development, regulatory approval, and/or commercialization; the potential for our programs and prospects to be negatively impacted by developments relating to our competitors, including the results of studies or regulatory determinations relating to our competitors; unfavorable results from preclinical studies; we may not realize the benefits (i) associated with orphan drug designation, including that orphan drug exclusivity may not effectively protect a product from competition and that such exclusivity may not be maintained or (ii) from the rare pediatric disease designation, including potential to receive a Priority Review Voucher (PRV) or derive any value therefrom; regulatory developments in the United States and foreign countries; our ability to obtain and maintain intellectual property protection for our product candidates and proprietary technologies; we may use our capital resources sooner than we expect; unstable market and economic conditions may adversely affect our business and financial condition and the broader economy and biotechnology industry; and other risks described in our prior filings with the Securities and Exchange Commission (SEC), including under the heading “Risk Factors” in our annual report on Form 10-K and any subsequent filings with the SEC. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof, and we undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date hereof. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. These and other factors could cause results to differ materially from those expressed in the estimates made by the independent parties and by us.

Here's a snapshot of TYRA

Differentiated assets

Precision small molecules targeting large opportunities in FGFR biology

Lead asset: TYRA-300, the first oral FGFR3-selective inhibitor in the clinic

Accelerated design

SNAP CHEMISTRY DESIGN

NASDAQ: TYRA

CASH Mar 31, 2024: $382.5M

RECENT PROGRESS

TYRA-300^{ACH}: on track to submit IND in 2H24

TYRA-300^{ONC}: SURF-301 Part B ongoing to support future NMIBC & mUC studies

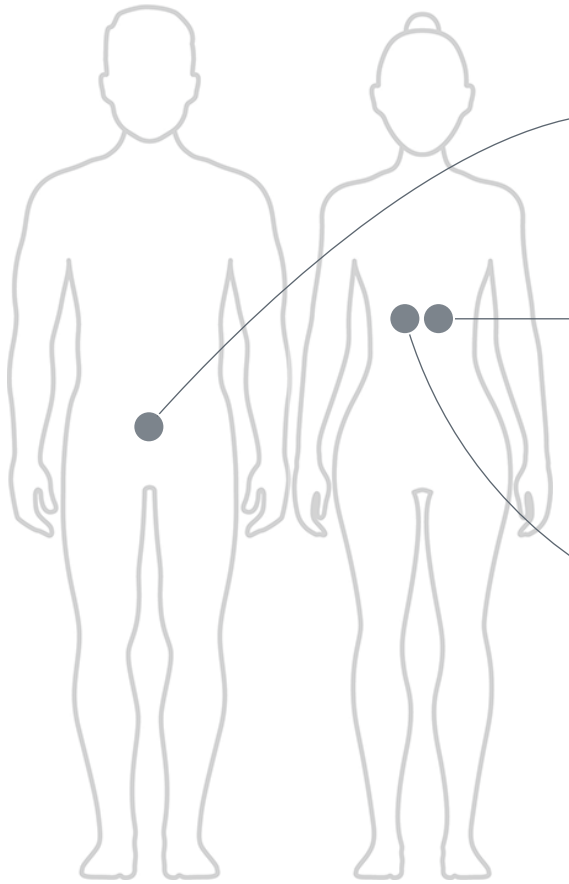
TYRA-430^{ONC}: completing IND-enabling studies

Strengthened Board with addition of Dr. Susan Moran & Dr. S. Michael Rothenberg

* ACH: Achondroplasia; ONC: Oncology
All TYRA small molecule inhibitors are for investigational use only

FGFR alterations are implicated in many cancers

FGFR1
2
3
4



UROTHELIAL CARCINOMA (UC)
~50% FGFR3 | ~40,000/yr (US)

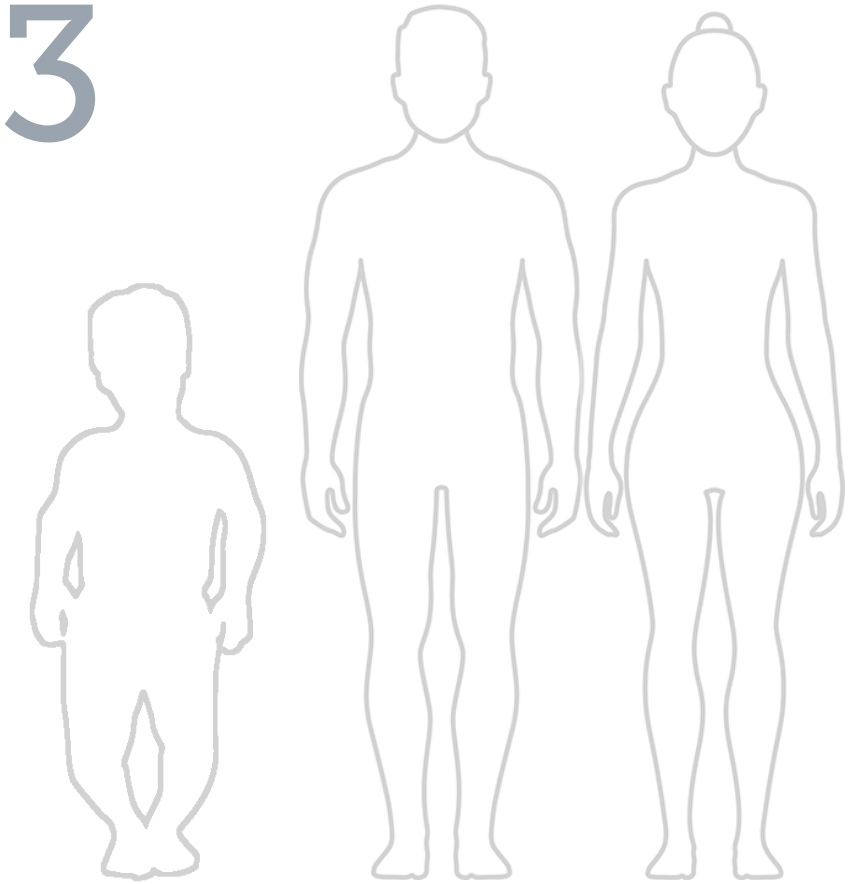
HEPATOCELLULAR CARCINOMA (HCC)
~30% FGFR4/3[1] | ~9,000/yr (US)

INTRAHEPATIC CHOLANGIOCARCINOMA (ICC)
~10–20% FGFR2 | ~1,700/yr (US)

Oncology figures represent 2022 US incidence across all stages of the disease
1. Estimate of FGF19 IHC+ in HCC; Kim, 2019

FGFR3 drives two large market opportunities

FGFR3



ACHONDROPLASIA (ACH)
~99% FGFR3 | ~3,000/yr (US)

OTHER FGFR3-RELATED SKELETAL DYSPLASIAS
~40,000/yr (US)

UROTHELIAL CARCINOMA (UC)
~50% FGFR3 | ~40,000/yr (US)

TYRA-300

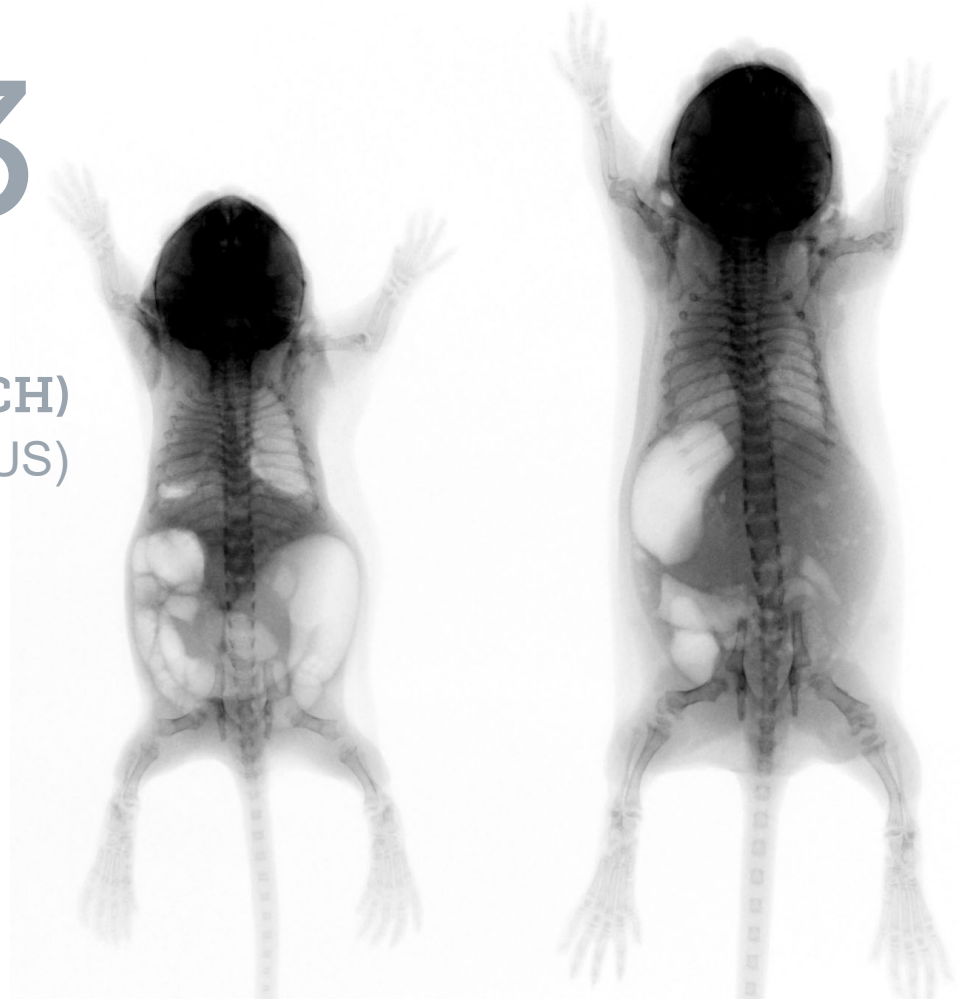
Has the potential to address these indications

Oncology figures represent 2022 US incidence across all stages of the disease; skeletal dysplasias represent 2022 US pediatric prevalence
TYRA-300 is an investigational product currently in a Phase 1 clinical trial for oncology and has not been approved by any governmental body for any therapy or indication

Pre-clinical studies with TYRA-300 produced encouraging results

FGFR3

ACHONDROPLASIA (ACH)
~99% FGFR3 | ~3,000/yr (US)



TYRA-300

17.9%

Observed increase in naso-anal length relative to vehicle*

*p<0.0001

Experiment conducted in the lab of Laurence Legeai-Mallet at Imagine Institute in Paris, France; Daily SQ injection of TYRA-300 at 1.2 mg/kg/day for 15 days starting at Day 1; *p<0.0001 n=8 for TYRA-300, after excluding two mice from dataset when molecular analysis showed chimeric incorporation of mutation, and n=10 for vehicle, after excluding one vehicle mouse when molecular analysis showed chimeric incorporation of mutation

Our expertise in FGFR biology creates a differentiated pipeline

GENETIC CONDITIONS	Estimated Annual US Addressable[1]	Discovery	IND-Enabling	Phase 1	Phase 2	Phase 3	Anticipated Milestone
FGFR3^{ACH}: TYRA-300	~3K	———	●·····	*Data from FGFR3^{ONC}*	····▶		Submit IND 2H '24

Potentially leading to additional skeletal dysplasias, including FGFR3-related conditions (HCH, SHOX), and pediatric short stature

ONCOLOGY							
FGFR3^{ONC}: TYRA-300	~40K	———	———	——— SURF301			Initial Ph1 data in 2H '24
FGFR2^{ONC}: TYRA-200	~5.5K	———	———	——— SURF201			Complete Ph1
FGFR4/3^{ONC}: TYRA-430	~9K	———	●———				Complete IND enabling

TYRA retains an active FGFR3 discovery program.

1. Represents FGFR3/FGFR2/FGF19+ incidence and relapses for TYRA300/200/430, prevalence for ACH

TYRA

TYRA-300^{ACH}

TYRA-300^{ONC}

SNÅP

TYRA

TYRA-300^{ACH}

TYRA-300^{ONC}

SNÅP

Potentially efficient development path in an attractive, rapidly-emerging market

ACH can result in serious clinical complications

ACH is the most common cause of disproportionate short stature

MECHANISM

FGFR3 G380R gain of function mutation accounts for ~99% of ACH[1,2]

FGFR3 inhibits chondrocyte proliferation and differentiation, resulting in decreased longitudinal bone growth[2]

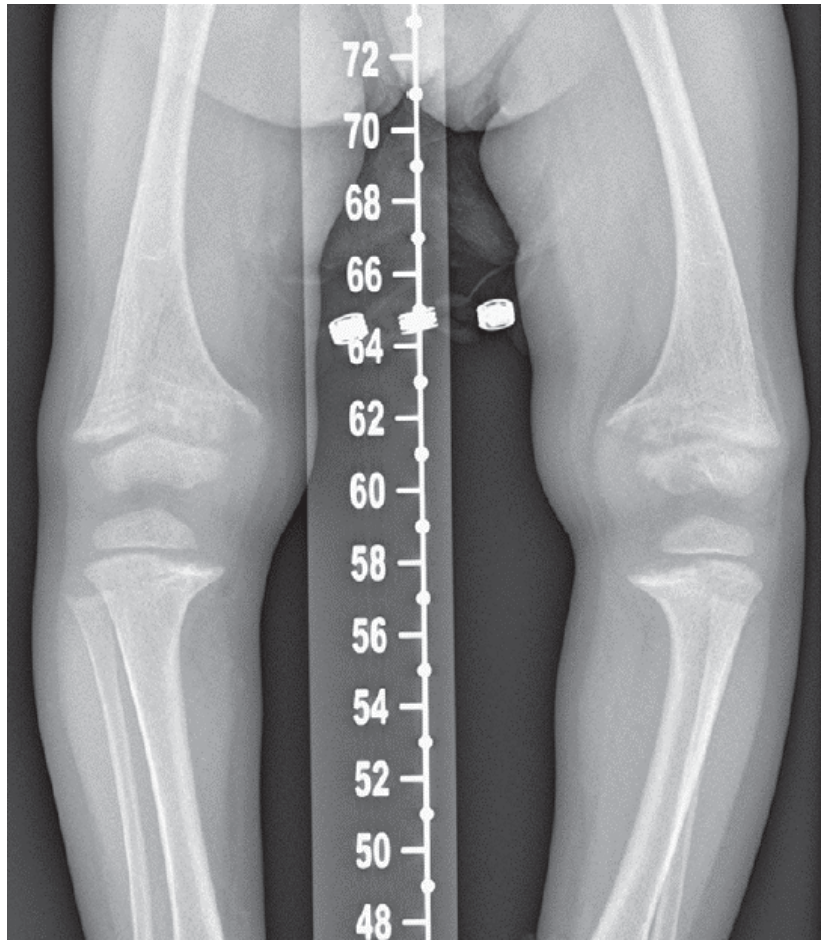
COMPLICATIONS

Infants can face serious complications related to critical foramen magnum stenosis[1,3]

Additionally: Pain, multiple surgeries, and functional limitations (e.g., reach, stride)

1. Pauli et al., 2019; 2. Ornitz and Legeai-Mallet, 2017; 3. Hecht et al., 1987

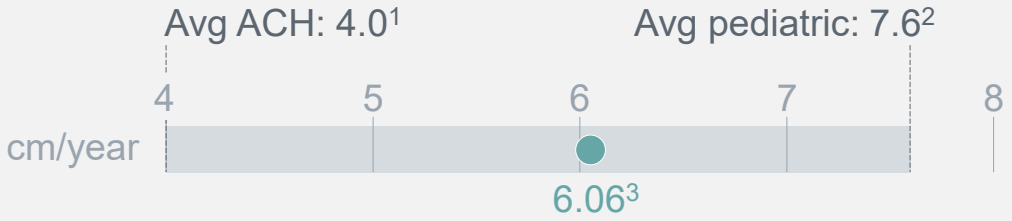
There is a strong need for an oral therapy selective for FGFR3



Vosoritide
(CNP analog)

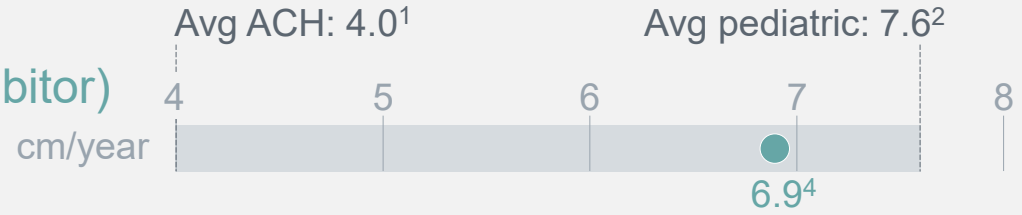
Daily injection,
US FDA-approved

Annual Height Velocity (AHV)



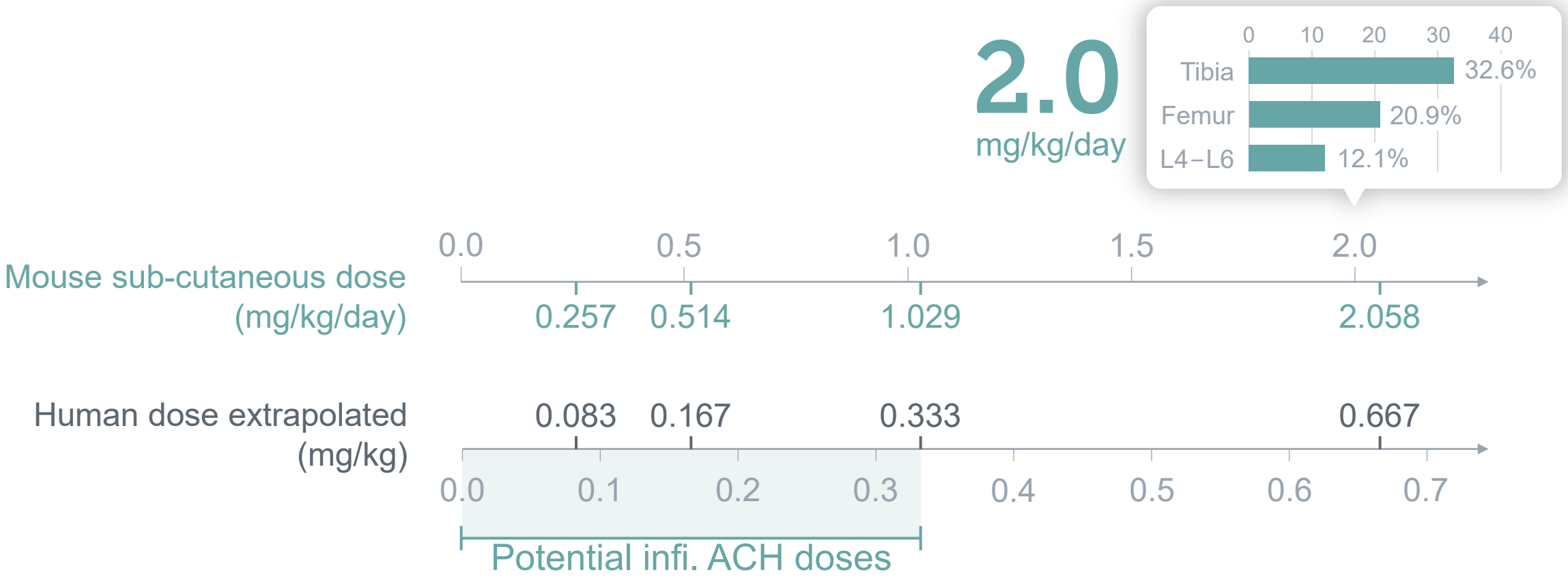
Low dose infigratinib
(Pan-FGFR1/2/3 inhibitor)

Oral, Phase 3



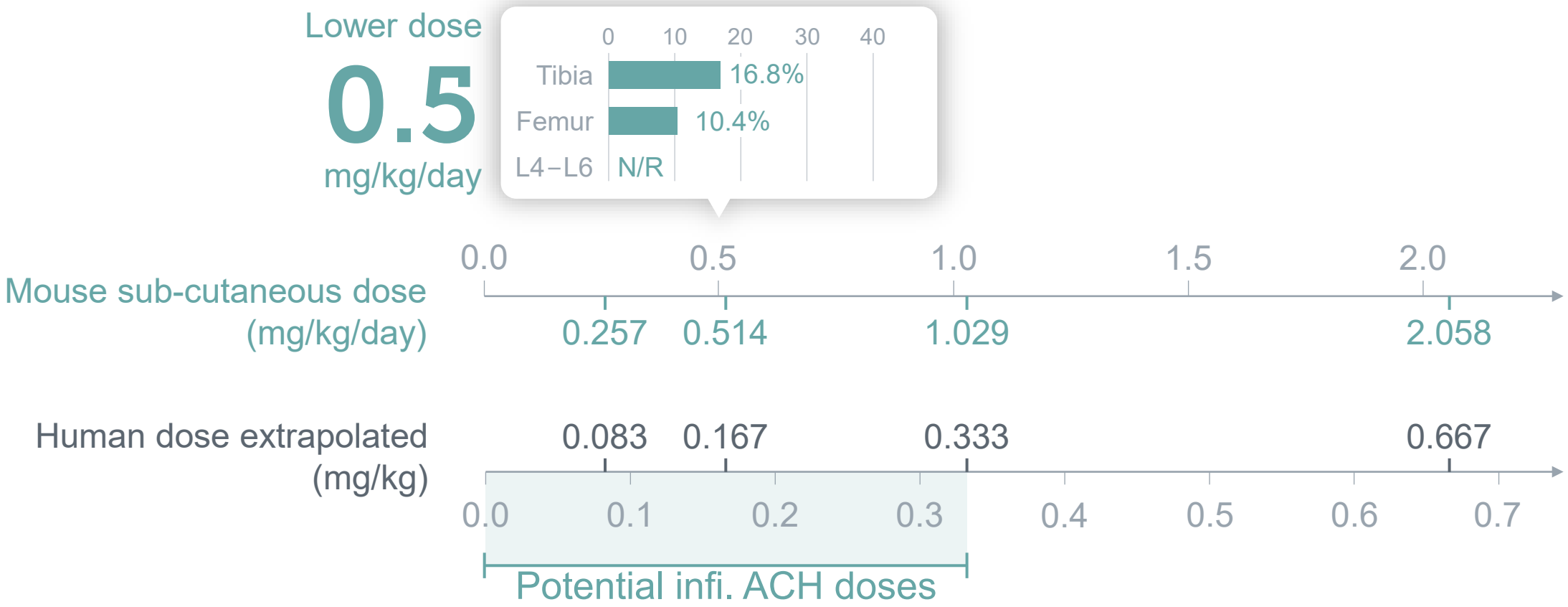
1. Savarirayan, 2021 (5 to 14yrs); 2. Merck Manuals (12mo to 10yrs); 3. Phase 2 Cohort 3 Data, VOXZOGO Label, Savarirayan, 2021;
4. Phase 2 Cohort 5 0.250mg/kg daily, Savarirayan, 2023 (ENDO)

2mg/kg/day infigratinib promoted growth in an ACH mouse model



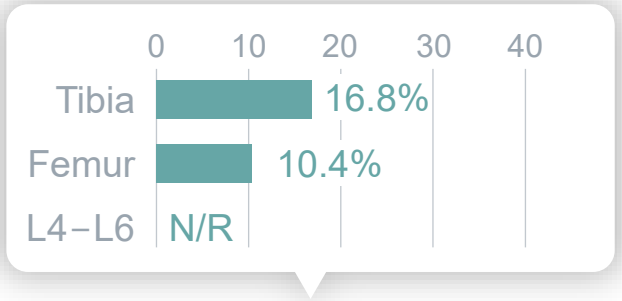
2.0mg/kg data in Komla-Ebri D, et al. J Clin Invest 2016; Potential infigratinib ACH doses and mouse to human dose extrapolation highlighted in Demuynck et. al. (ASHG, 2019)

Subsequent 0.5mg/kg/day infigratinib dose demonstrated less growth



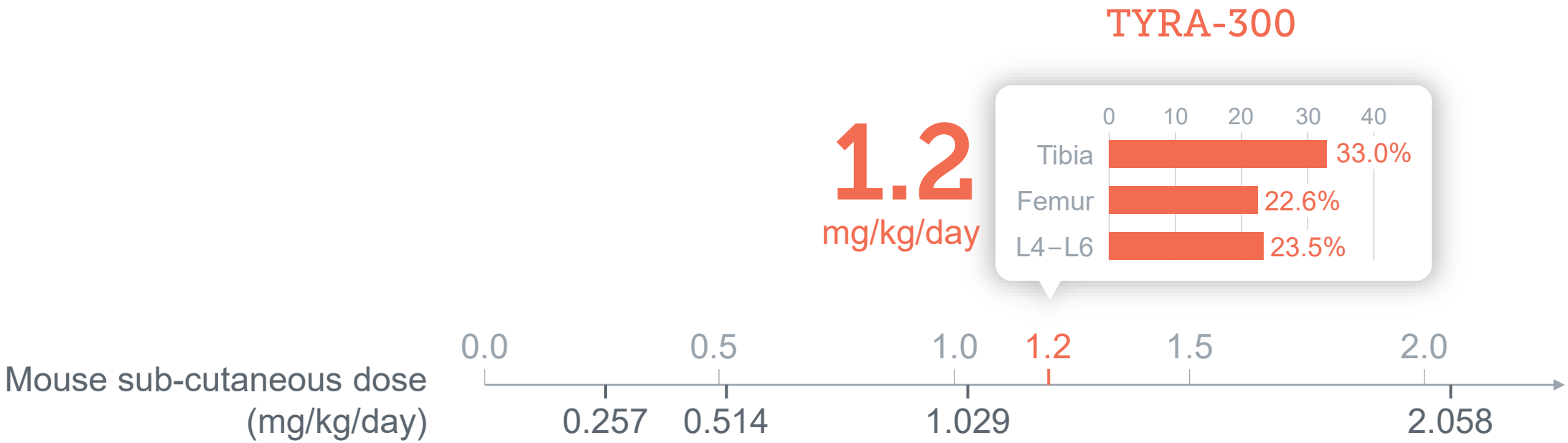
Demuynck et. al. (ASHG, 2019)

0.5mg/kg/day infigratinib mouse dose translates closer to Ph3 dose



Demuynck et. al. (ASHG, 2019)

TYRA-300 was highly active in a similar ACH pre-clinical study



Data generated from an experiment conducted in the lab of Dr. Laurence Legeai-Malei

Pan-FGFR inhibitors have exhibited toxicity in oncology

FGFR1: HYPERPHOSPHATEMIA

PAN-FGFR INHIBITORS	% PATIENTS AFFECTED
PEMAZYRE (pemigatinib[1])	60%
LYTGOBI (futibatinib[2])	88%
BALVERSA (erdafitinib[2])	76%
TRUSELTIQ (infigratinib[1])	82%

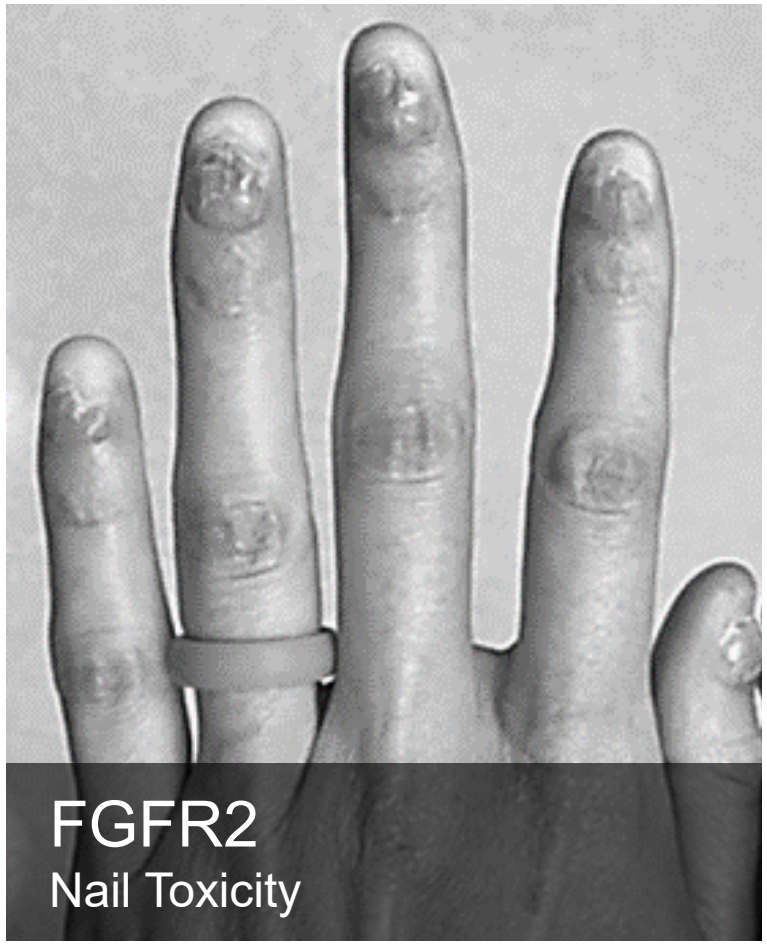
FGFR1
Hyperphosphatemia

1. FGFR1-3 inhibitor 2. FGFR1-4 inhibitor
Source: PEMAZYRE (pemigatinib) label, FIGHT-202 data, accessed 04/15/24; LYTGOBI (futibatinib) label, pooled data, accessed 04/15/24; BALVERSA (erdafitinib) label, BLC3001 Cohort 1 data, accessed 04/15/24; TRUSELTIQ (infigratinib) label, pooled data, accessed 04/15/24; Chan, 2022; Kim, 2019; Hollebecque, 2022 (ESMO)

Pan-FGFR inhibitors have exhibited toxicity in oncology

OTHER TOXICITIES

FGFR1
Hyperphosphatemia

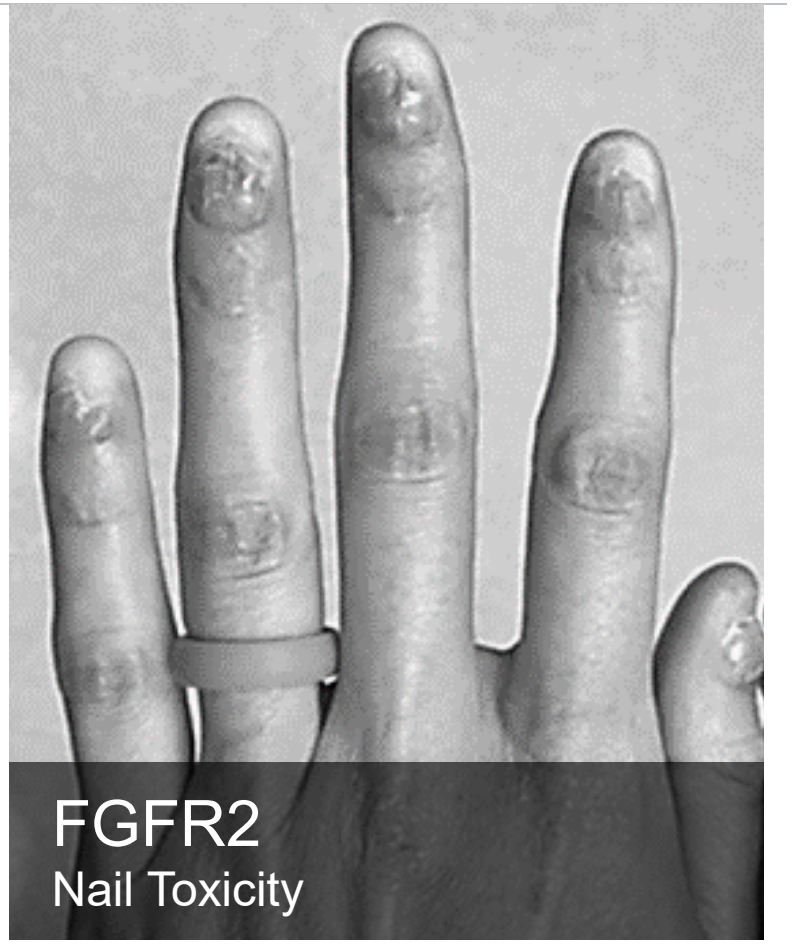


FGFR2
Nail Toxicity

Source: PEMAZYRE (pemigatinib) label, FIGHT-202 data, accessed 04/15/24; LYTGOBI (futibatinib) label, pooled data, accessed 04/15/24; BALVERSA (erdafitinib) label, BLC3001 Cohort 1 data, accessed 04/15/24; TRUSELTIQ (infigratinib) label, pooled data, accessed 04/15/24; Chan, 2022; Kim, 2019; Hollebecque, 2022 (ESMO)

Pan-FGFR inhibitors have exhibited toxicity in oncology

FGFR1
Hyperphosphatemia



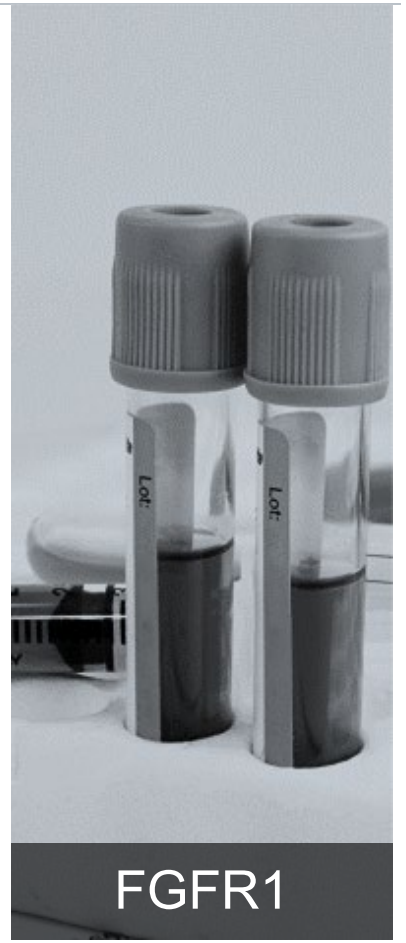
FGFR2
Nail Toxicity

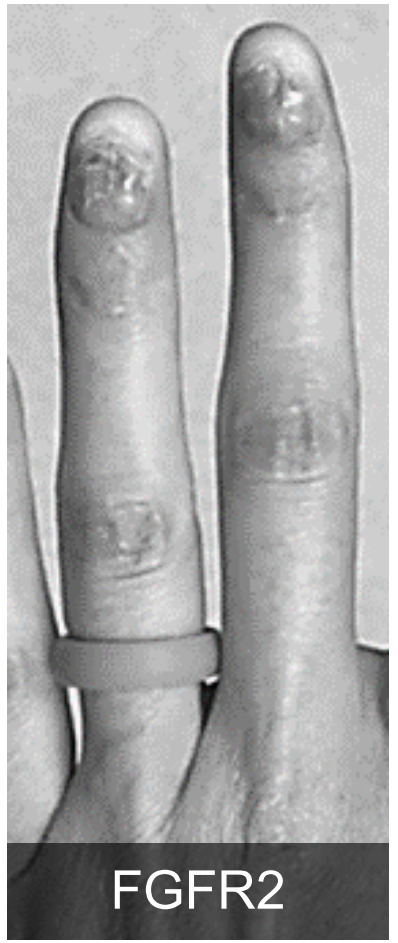
FGFR4
Diarrhea

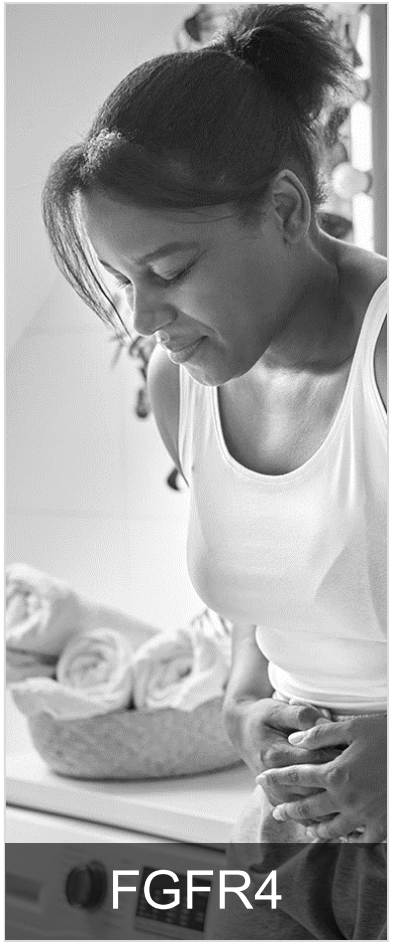
Source: PEMAZYRE (pemigatinib) label, FIGHT-202 data, accessed 04/15/24; LYTGOBI (futibatinib) label, pooled data, accessed 04/15/24; BALVERSA (erdafitinib) label, BLC3001 Cohort 1 data, accessed 04/15/24; TRUSELTIQ (infigratinib) label, pooled data, accessed 04/15/24; Chan, 2022; Kim, 2019; Hollebecque, 2022 (ESMO)

Pan-FGFRi side effects led to dose reductions & discontinuations

PAN-FGFR INHIBITOR	% PATIENTS DOSE REDUCTIONS & DISCONTINUATIONS
PEMAZYRE (pemigatinib[1])	23%
LYTGOBI (futibatinib[2])	63%
BALVERSA (erdafitinib[2])	83%
TRUSELTIQ (infigratinib[1])	75%



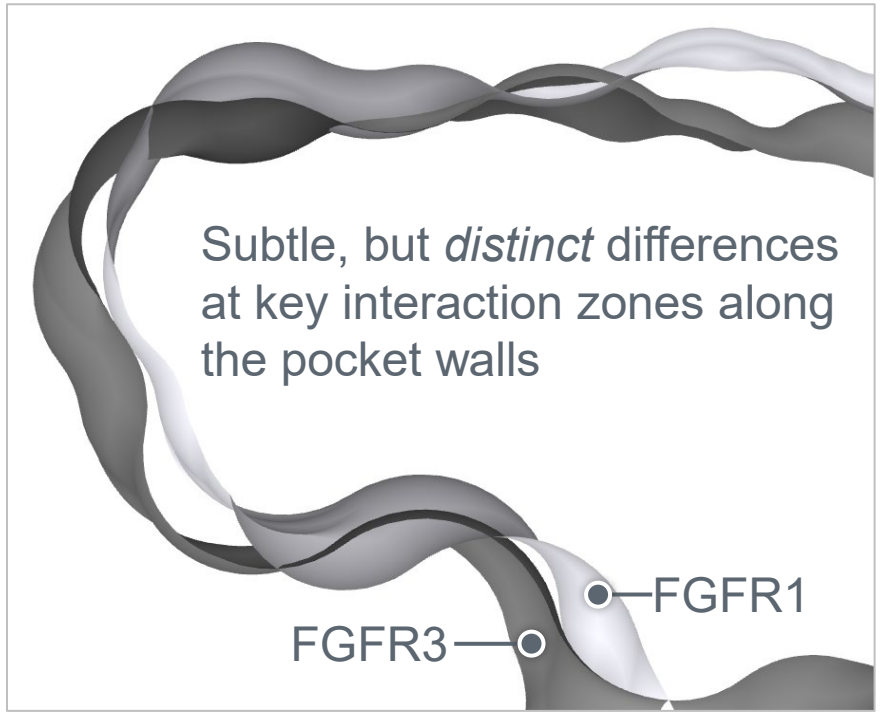




1. FGFR1-3 inhibitor 2. FGFR1-4 inhibitor
Source: PEMAZYRE (pemigatinib) label, FIGHT-202 data, accessed 04/15/24; LYTGOBI (futibatinib) label, pooled data, accessed 04/15/24; BALVERSA (erdafitinib) label, BLC3001 Cohort 1 data, accessed 04/15/24; TRUSELTIQ (infigratinib) label, pooled data, accessed 04/15/24; Chan, 2022; Kim, 2019; Hollebecque, 2022 (ESMO)

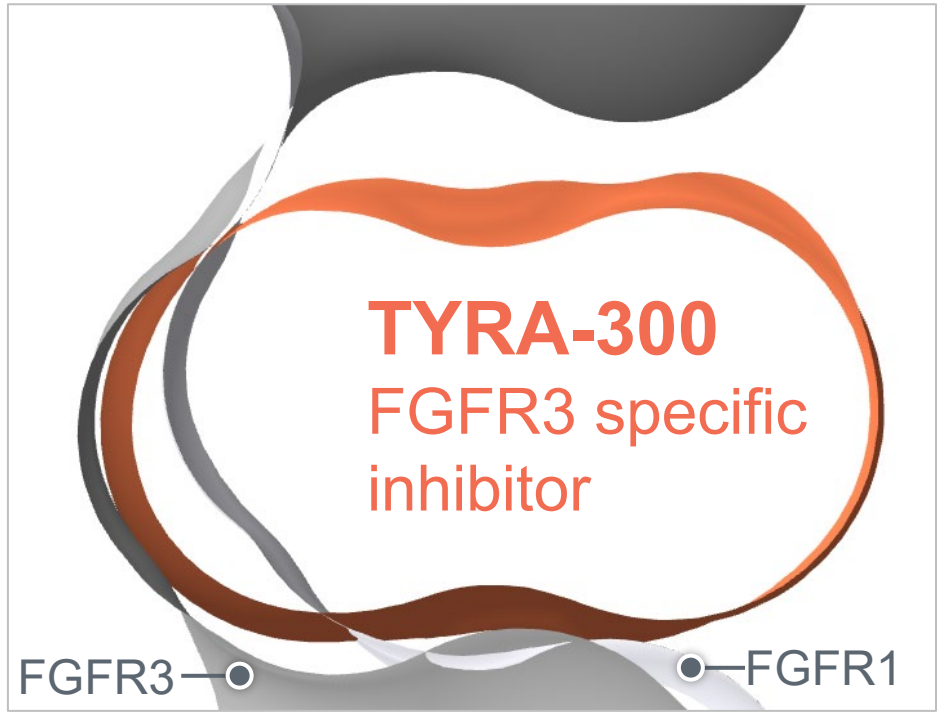
The challenge: FGFR family active sites are nearly identical

FGFR isoform selectivity



MOLECULAR MODEL

dozens of high-resolution crystal structures

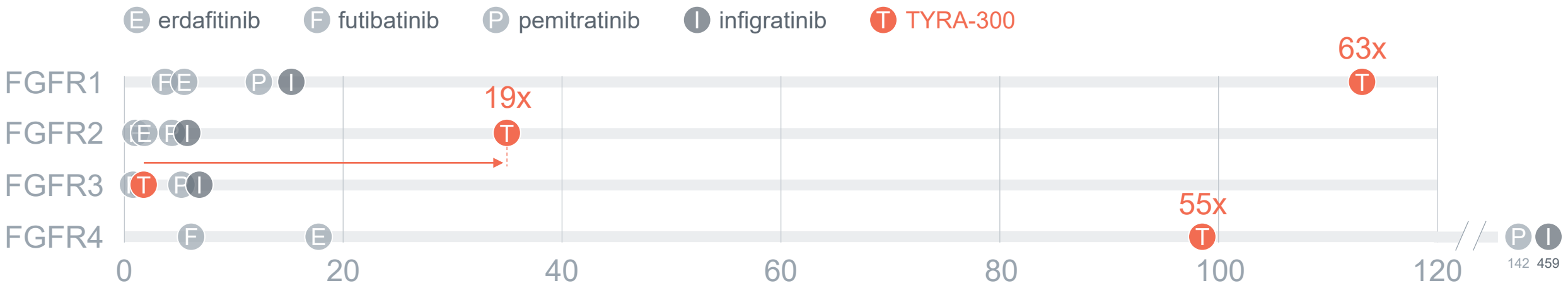


CRYSTALLOGRAPHY

3.0Å cross-sections

TYRA-300 was more selective for FGFR3 than pan-FGFR inhibitors

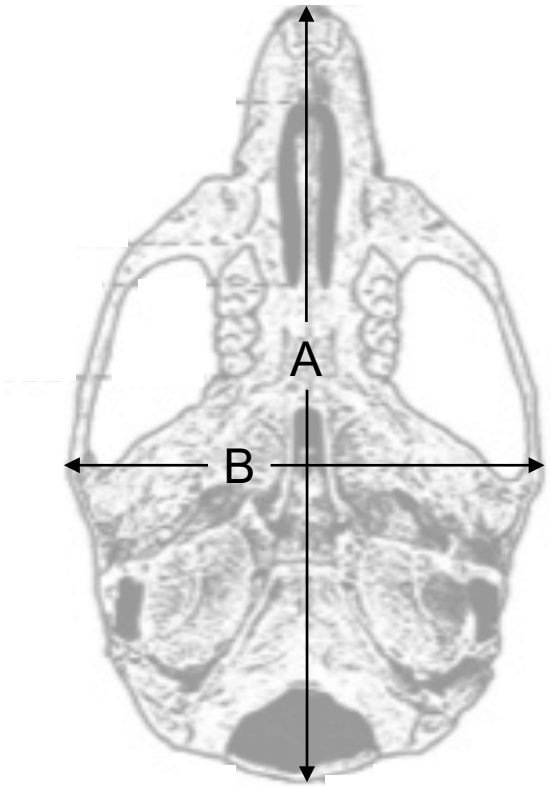
Selectivity observed for TYRA-300 vs. approved or late-stage clinical compounds: *in vitro* Ba/F3 Cellular IC_{50} (nM)

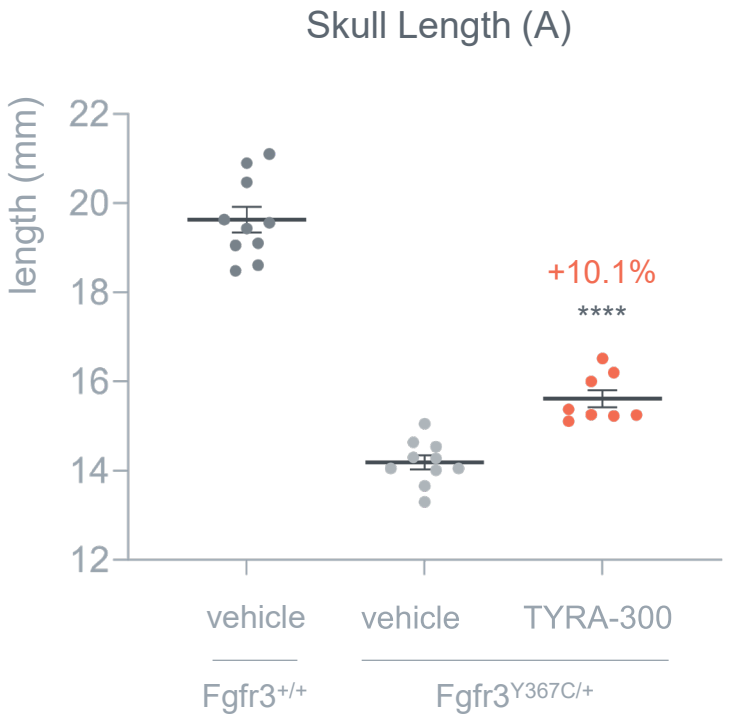


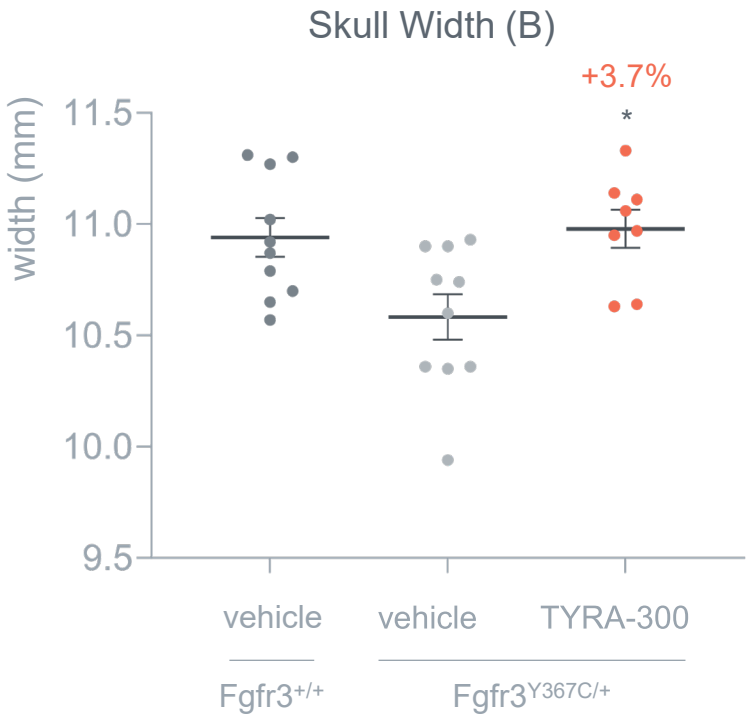
	E	F	P	I
FGFR1	4.2x	4.9x	2.4x	2.2x
FGFR2	1.4x	1.3x	0.8x	0.8x
FGFR4	14x	7.6x	27x	67x

All experiments conducted under identical conditions, tested in duplicate.

TYRA-300 improved the shape of the skull in ACH mouse model



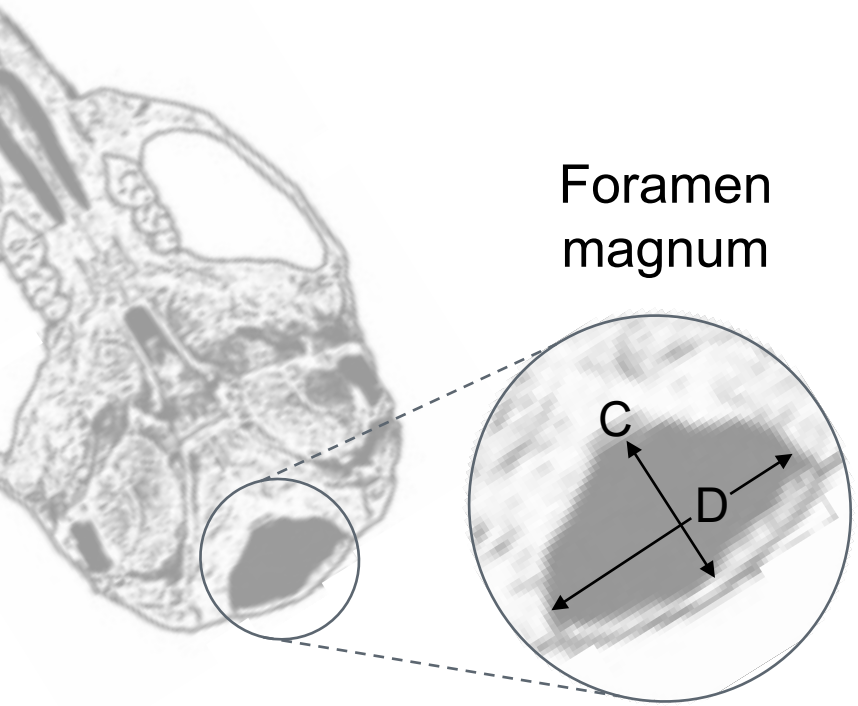


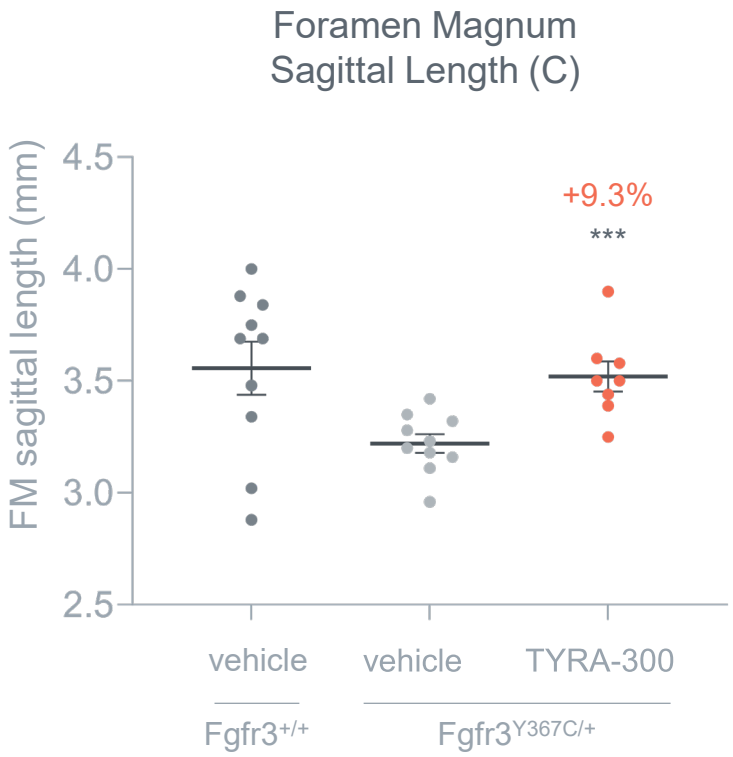


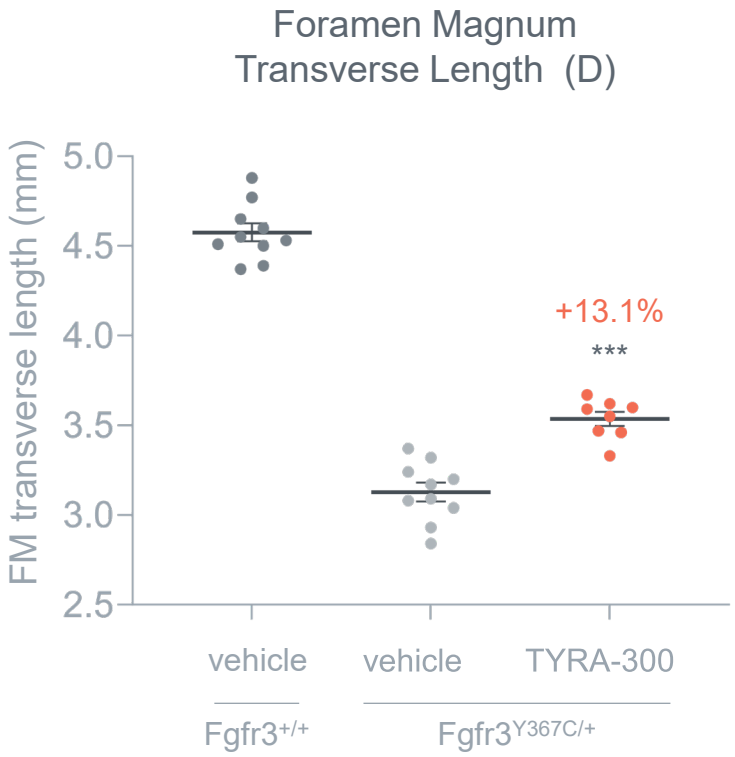
Mann Whitney test vs. vehicle
**** p < 0.0001 | * p < 0.05

Experiment conducted in the lab of Laurence Legeai-Mallet at Imagine Institute in Paris, France; Daily subcutaneous injection of TYRA-300 at 1.2 mg/kg/day for 15 days starting at Day 1; n=8 for TYRA-300 and n=10 for vehicle, after excluding three mice from dataset when molecular analysis showed chimeric incorporation of mutation.

TYRA-300 improved the foramen magnum in ACH mouse model







Mann Whitney test vs. vehicle
*** p < 0.001

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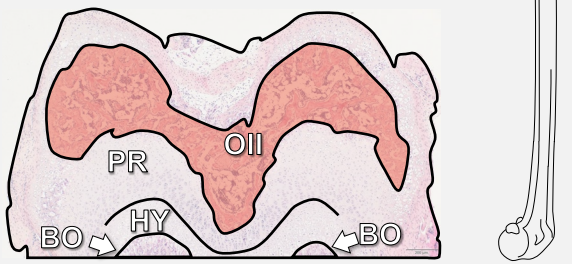
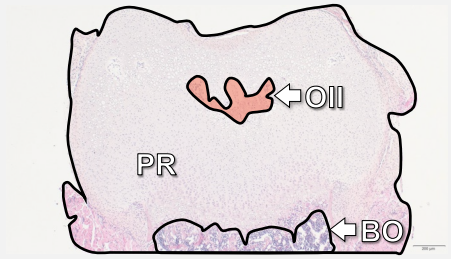
TYRA-300 restored growth plate architecture in ACH mouse model

A **Wild Type**
(normal FGFR3)

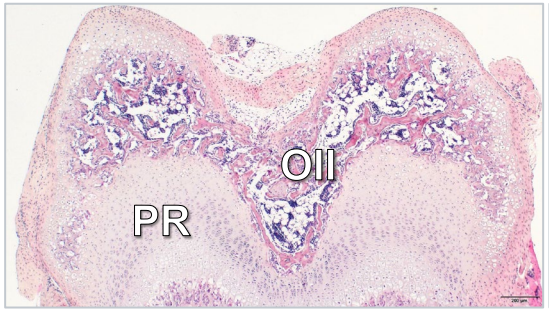


B **Mutant**
(activated FGFR3)

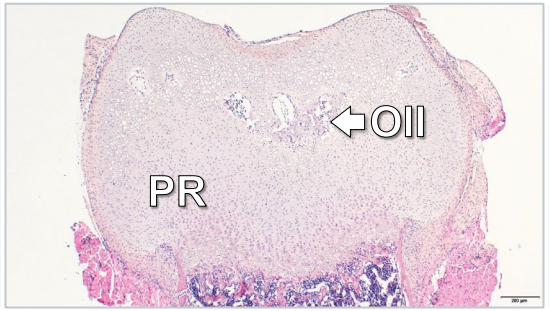


DISTAL FEMUR

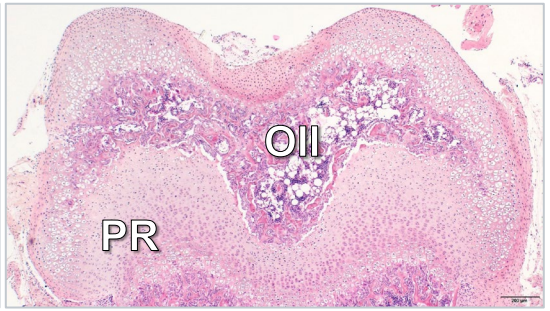
A $Fgfr3^{+/+}$
vehicle treated



B $Fgfr3^{Y367C/+}$
vehicle treated



$Fgfr3^{Y367C/+}$
TYRA-300 treated



Experiment conducted in the lab of Laurence Legeai-Mallet at Imagine Institute in Paris, France
Hematoxylin and eosin stains of distal femurs. PR: proliferating chondrocytes, OII: secondary ossification center, HY: hypertrophic chondrocytes, BO: bone

Our clinical strategy will focus on three critical areas

1. The ACH community's needs
2. FDA considerations
3. Potential differentiation

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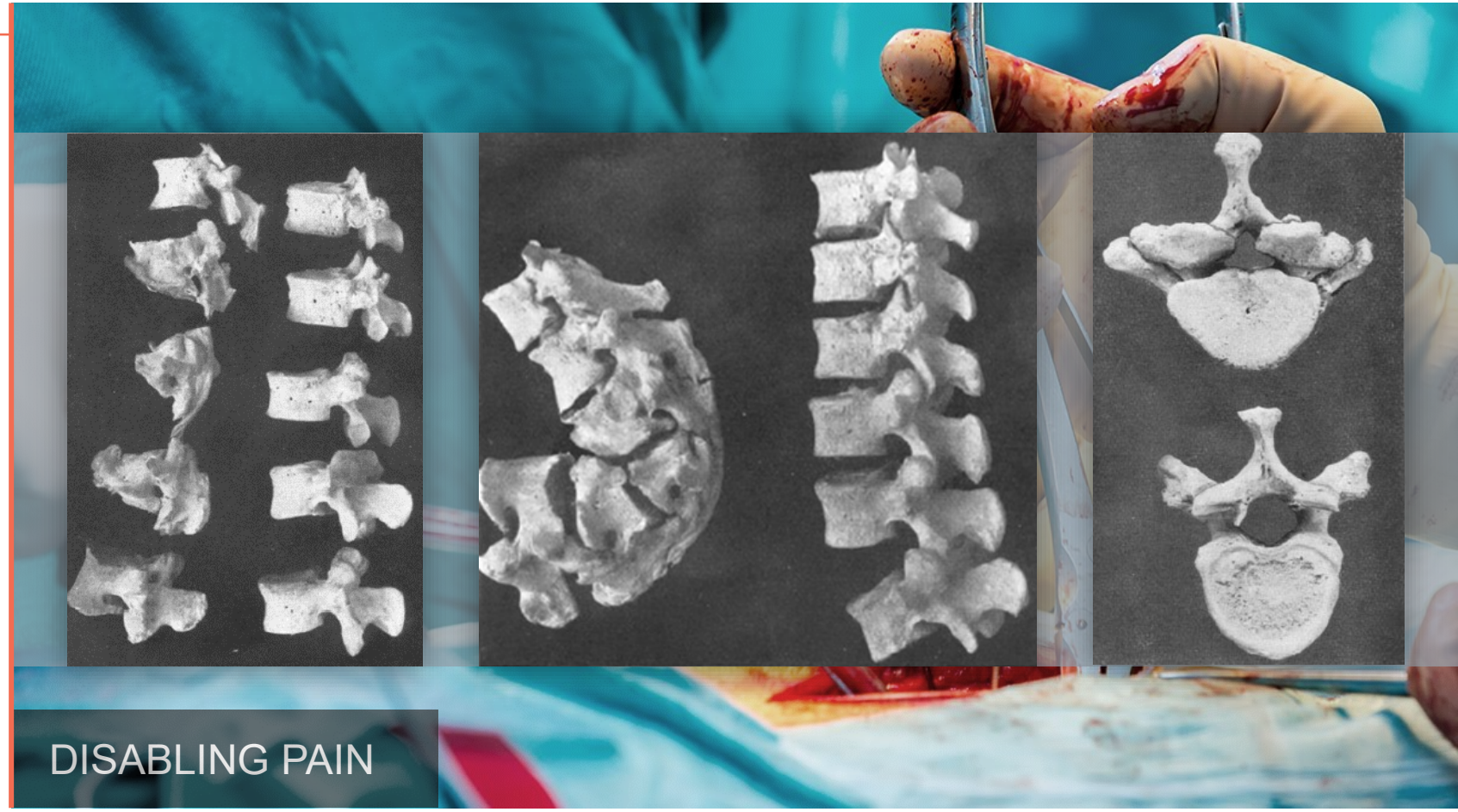
2. FDA considerations
3. Potential differentiation



Our clinical strategy will focus on three critical areas

1. The ACH community's needs
2. FDA considerations
3. Potential differentiation

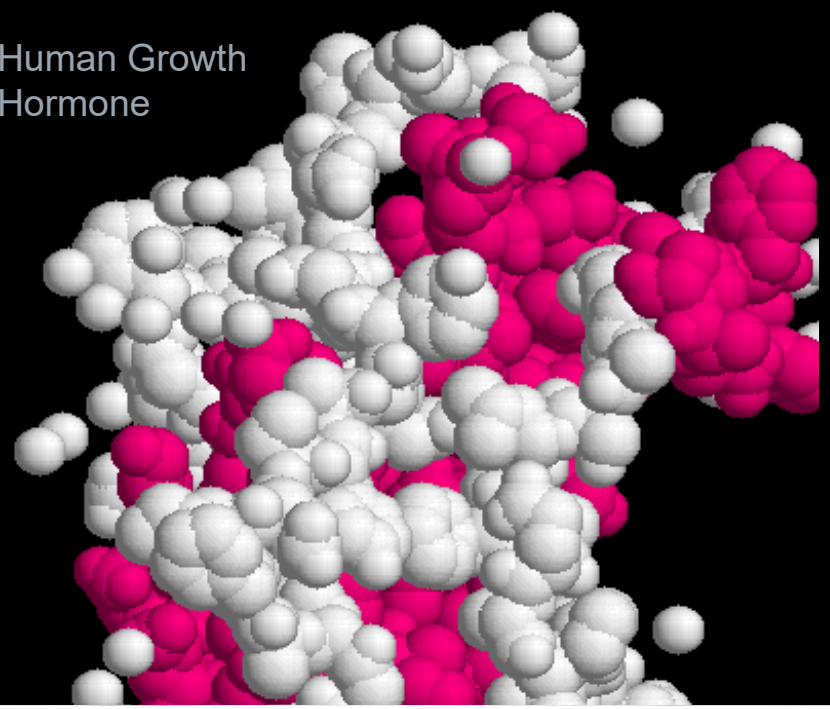
CURRENT VIEW

No validated growth prediction models link short-term AHV with final height.

Co-evaluation of other clinically meaningful endpoints is recommended.

HISTORICAL CONTEXT

Human Growth Hormone



Our proposed development plan sets up potential for differentiation

1. The ACH community's needs
2. FDA considerations
3. Potential differentiation

	PHASE 2 and Pivotal Randomized, multi-dose with control[1,2]	Long Term Follow Up Potential comparative cohorts
AHV	Surrogate, 6, 12 & 24 mo	Potential differentiation
Functional improvements	Exploratory endpoints	
Clinical sequalae	Exploratory endpoints	
Final height	Not previously required for accelerated approval[3]	Previously required for full approval[3]

1. Initially with 5-12yrs naïve, then expanding 2+yrs and 5-12 treatment experienced; 2. Based on currently available information, including current treatment landscape, and subject to change based on clinical results, regulatory interactions and other factors; 3. Based on VOXZOGO precedent

We plan to design our Ph2 to determine recommended dose(s)

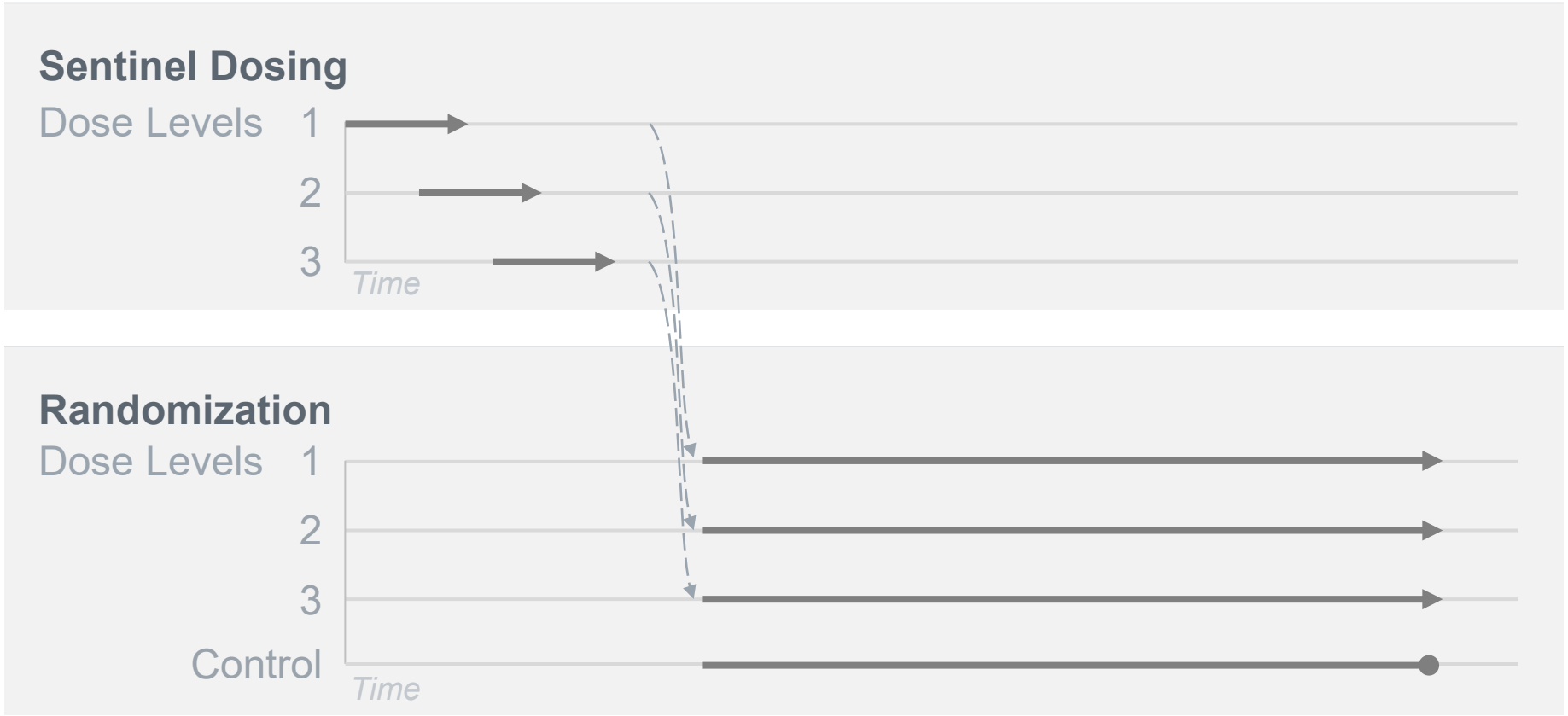
COHORT 1: ACH CHILDREN AGES 5-12[1]

Illustrative

Are target exposures achieved?

Any safety signals?

What is the recommended dose?



1. Initially with 5-12yrs naïve, then expanding 2+yrs and 5-12 treatment experienced

Based on currently available information, including current treatment landscape, and subject to change based on clinical results, regulatory interactions and other factors

There are multiple development opportunities beyond ACH

FGFR3 GERMLINE MUTATIONS

SKELETAL DYSPLASIAS

- Achondroplasia (~3K) ← *ODD and RPD granted*
- Hypochondroplasia (~2K)
- Thanatophoric dysplasia
- SADDAN syndrome
- Double dominant ACH

CRANIOSYNOSTOSIS

- Crouzon syndrome with acanthosis nigricans
- Muenke syndrome

OTHER GENETIC SHORT STATURE

- Leri-Weill dyschondrosteosis (~26K)
- Turner syndrome (~10K)
- Osteogenesis imperfecta (~4K)
- Mucopolysaccharidoses IVA and VI
- Laron syndrome (Growth Hormone Insensitivity)

PEDIATRIC SHORT STATURE

- Idiopathic short stature (~700K[1])

Addressable US pediatric population; Source: company research
1. Represents children ages 4-17 under 2.25 standard deviations from mean height

TYRA

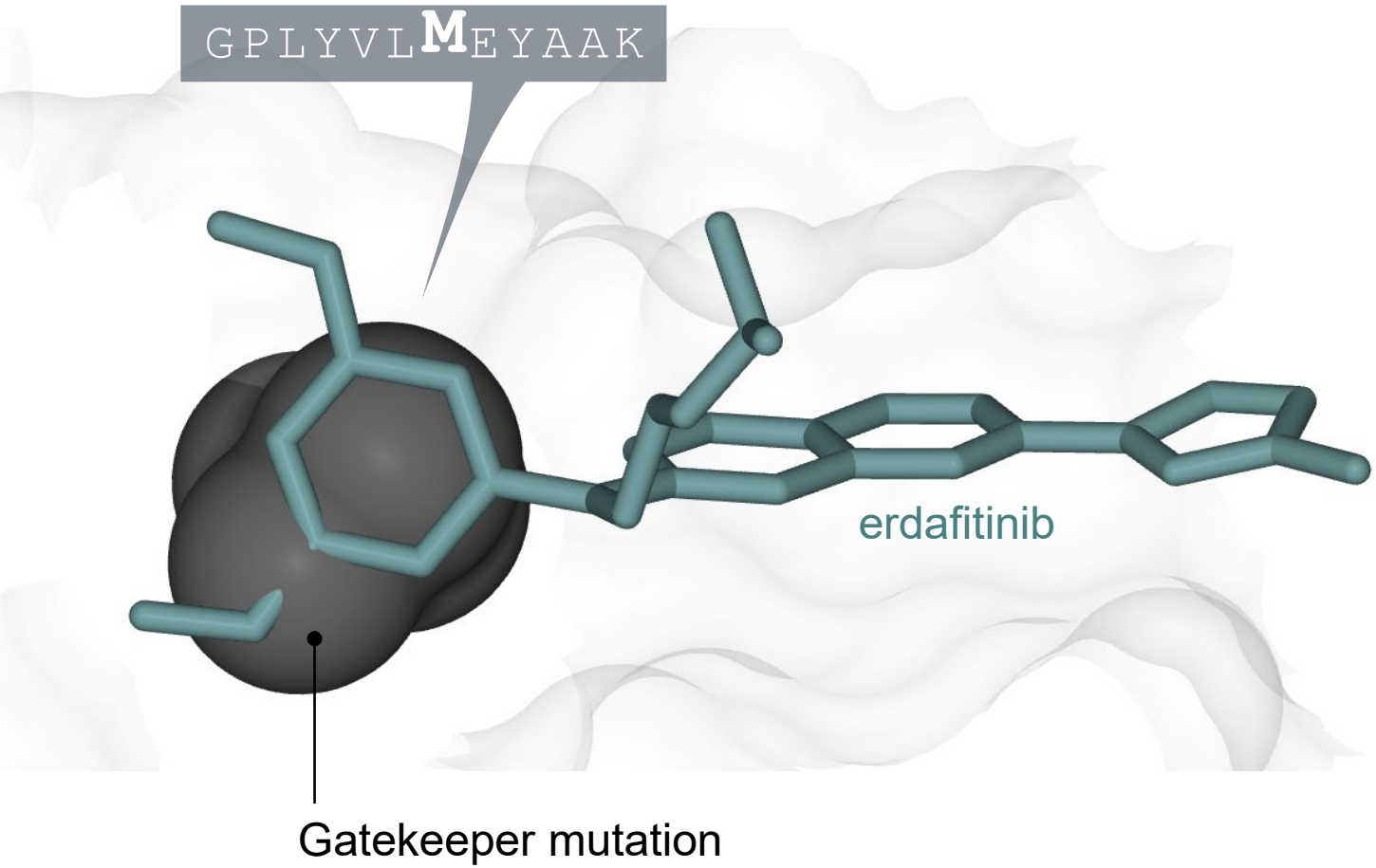
TYRA-300^{ACH}

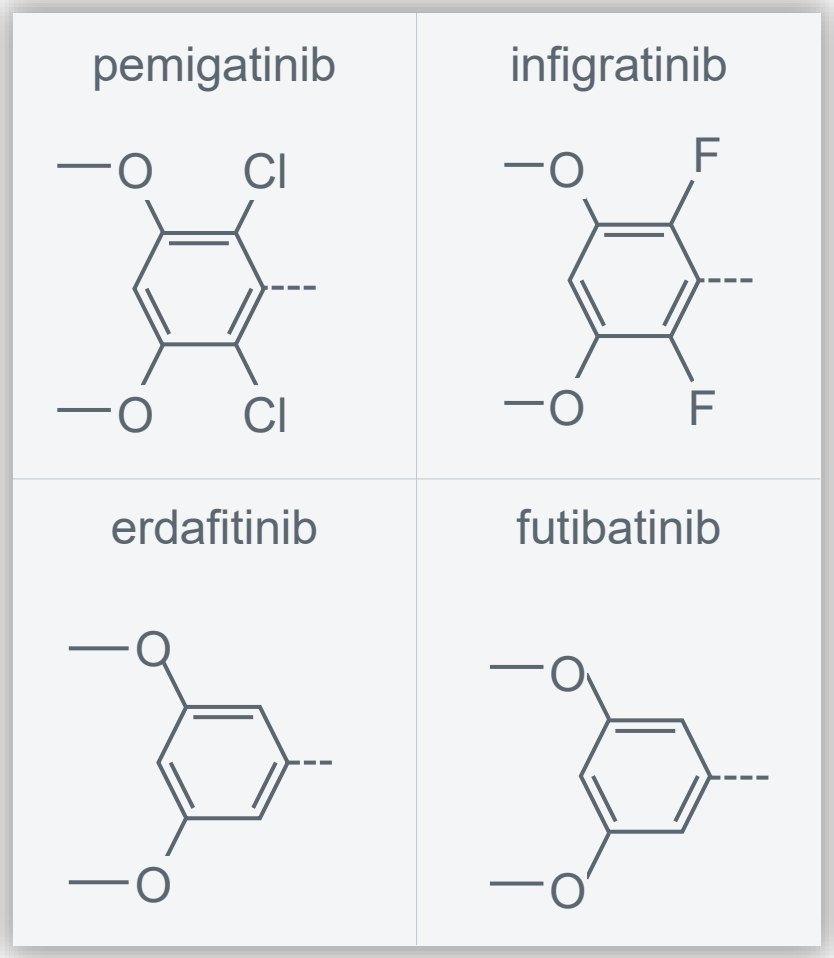
TYRA-300^{ONC} — Designed to potentially improve tolerability and durability over current standards of care

SNÅP

Resistance mutations potentially limit the durability of current drugs

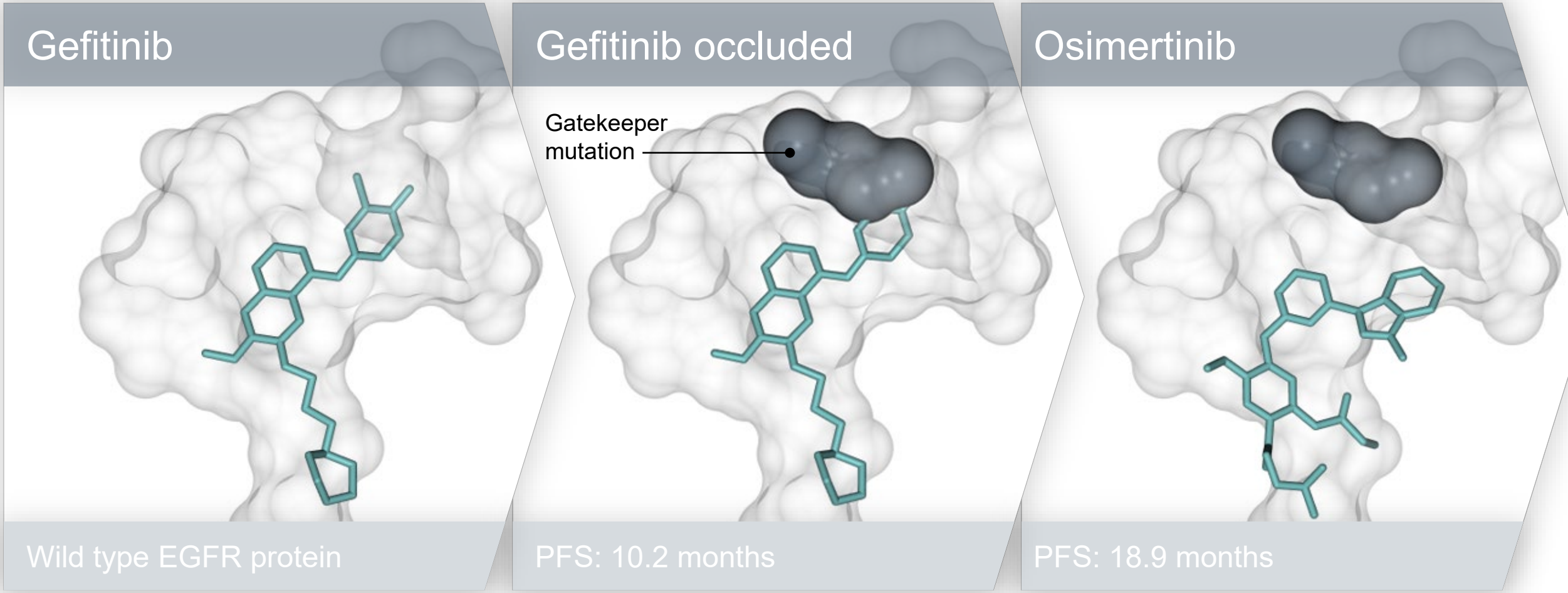
FGFR3 Example





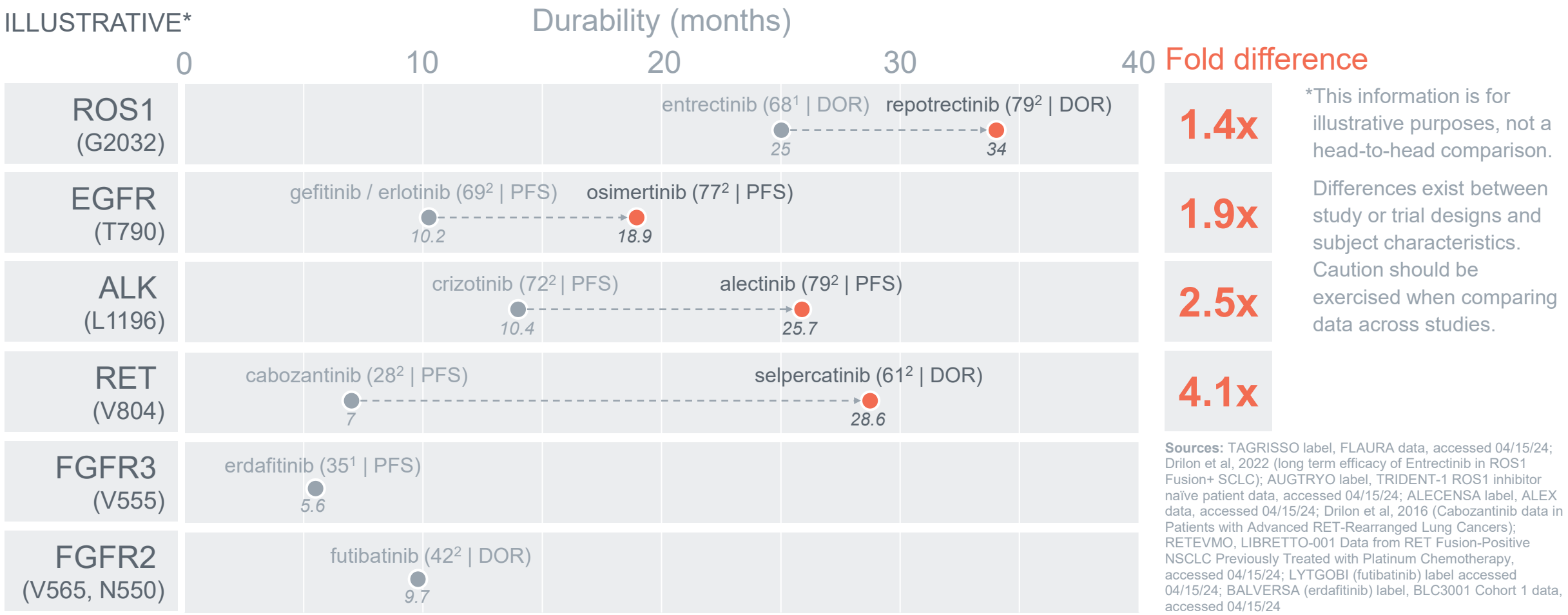
Structural insights provide a rational path to address recurrence

EGFR Example



Source: TAGRISSO label, FLAURA data, accessed 04/14/24; Gefitinib data represents data from FLAURA comparator arm of either gefitinib or erlotinib

Next gen drugs extend progression free survival



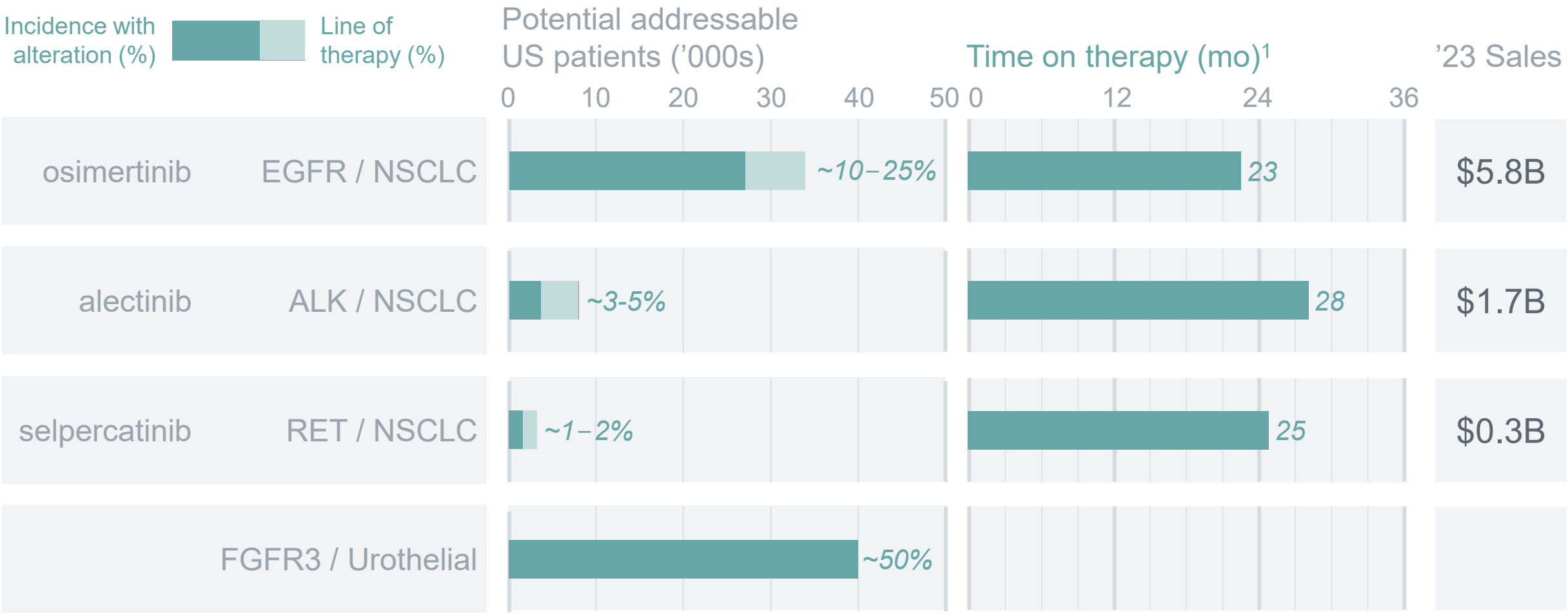
*This information is for illustrative purposes, not a head-to-head comparison.

Differences exist between study or trial designs and subject characteristics. Caution should be exercised when comparing data across studies.

1. Objective Response Rate (%); 2. Overall Response Rate (%)

Sources: TAGRISSO label, FLAURA data, accessed 04/15/24; Drilon et al, 2022 (long term efficacy of Entrectinib in ROS1 Fusion+ SCLC); AUGTRYO label, TRIDENT-1 ROS1 inhibitor naïve patient data, accessed 04/15/24; ALECENSA label, ALEX data, accessed 04/15/24; Drilon et al, 2016 (Cabozantinib data in Patients with Advanced RET-Rearranged Lung Cancers); RETEVMO, LIBRETTO-001 Data from RET Fusion-Positive NSCLC Previously Treated with Platinum Chemotherapy, accessed 04/15/24; LYTGOBI (futibatinib) label accessed 04/15/24; BALVERSA (erdafitinib) label, BLC3001 Cohort 1 data, accessed 04/15/24

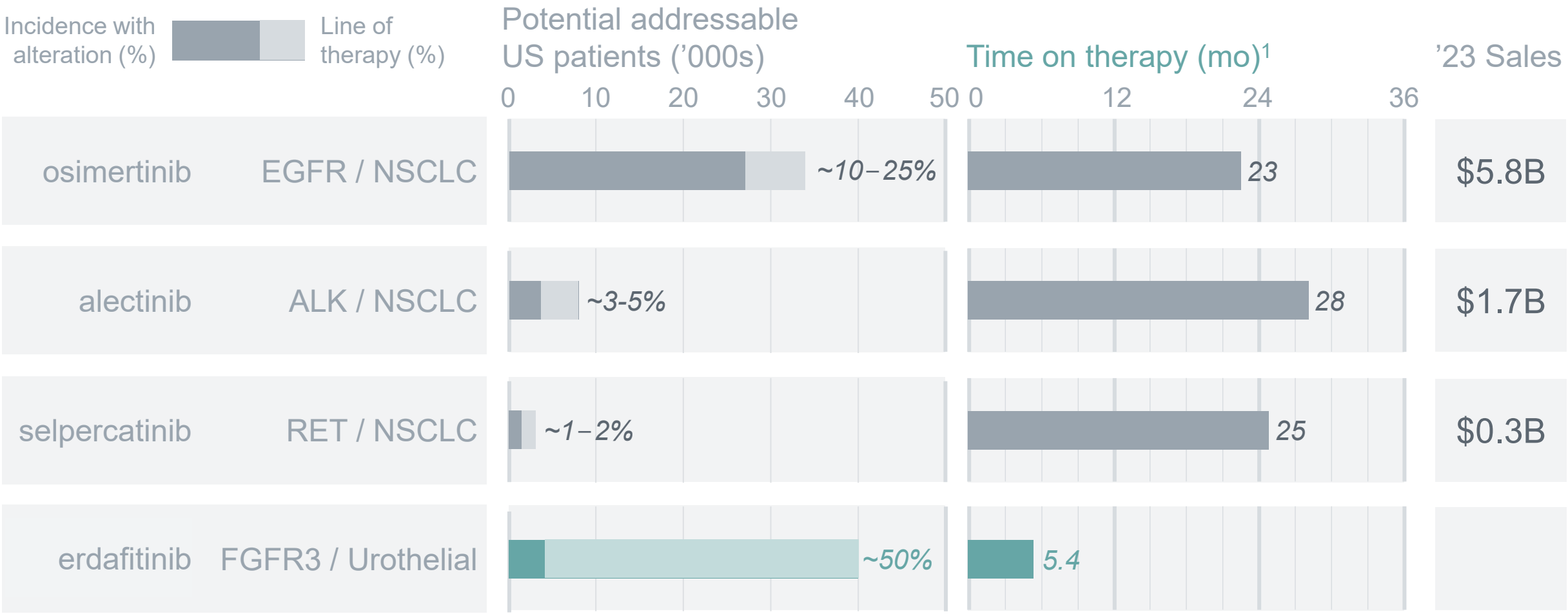
FGFR3 positive urothelial cancer is an outsized opportunity



1. Median duration of exposure for earliest line study

Source: SEER, ACS, NCCN; UpToDate; Company filings; GlobalData; Wu et al, 2020; Zhang et al, 2016; Graham et al., 2018; Mok et al, 2020; Subbiah et al, 2020; Drilon et al, 2023; Kacew et al, 2020; Matulewicz, 2020; Rizzo, 2022; Necchi, 2020

Despite a large opportunity, pan-FGFR inhibitors fall short



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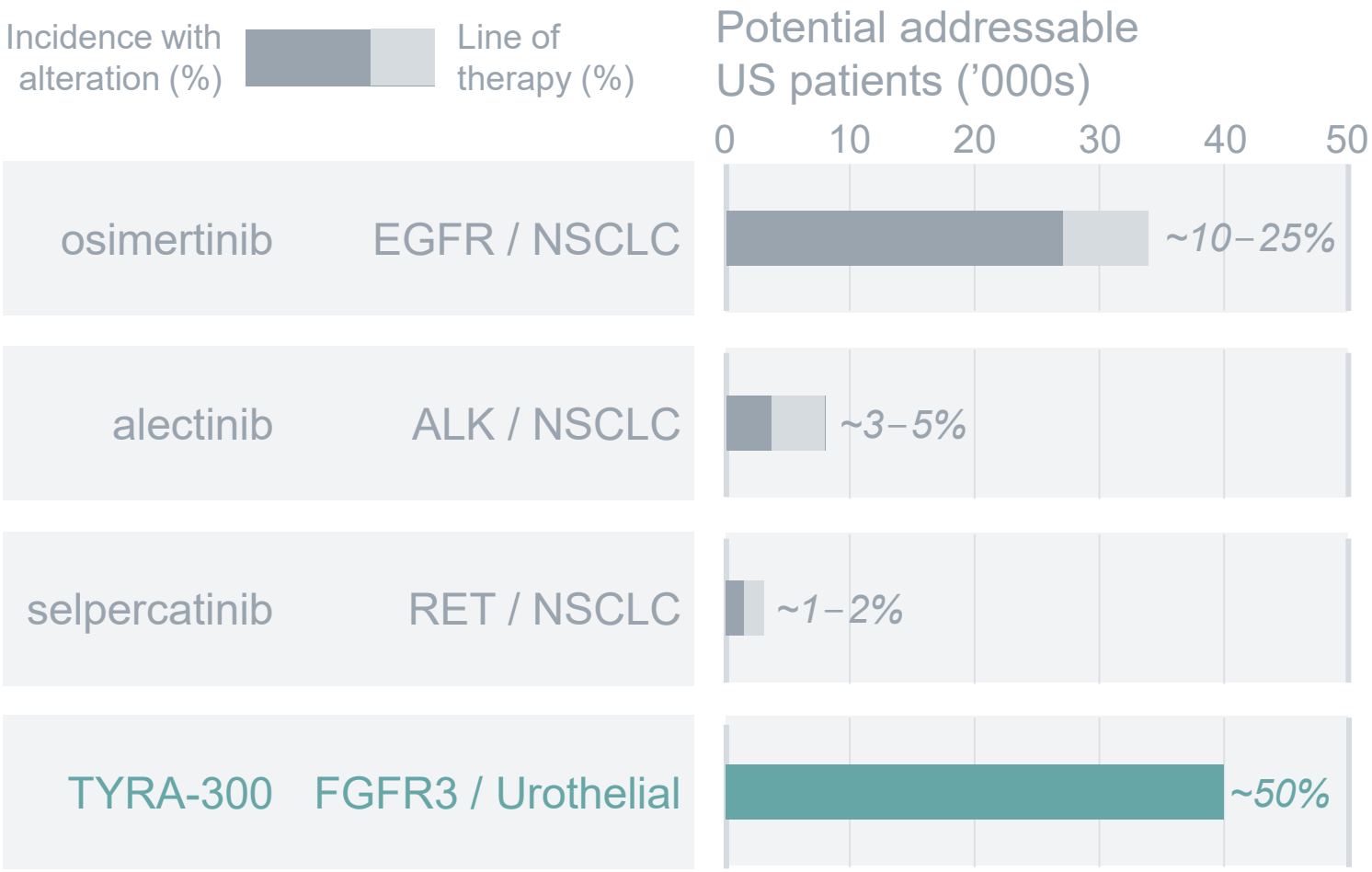
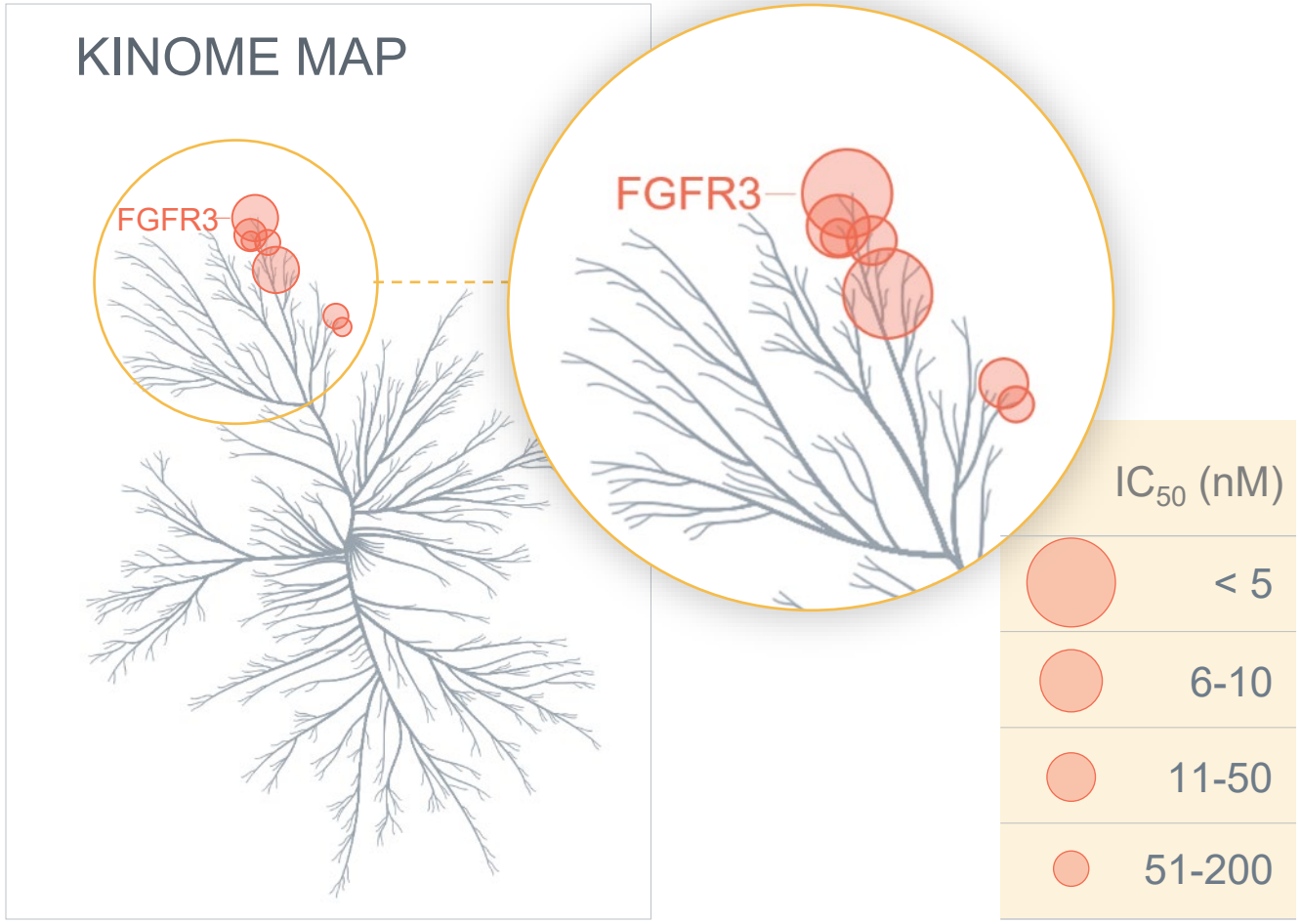
TYRA-300 is designed to address the unmet need



NEXT GEN IMPROVEMENT	
Mutation	**Selectivity**
Gatekeeper	Mutant selectivity
Gatekeeper	ALK selectivity
Gatekeeper	RET selectivity
Gatekeeper	FGFR isoform selectivity

Source: SEER, ACS, NCCN; UpToDate; Company filings; GlobalData; Wu et al, 2020; Zhang et al, 2016; Graham et al., 2018; Mok et al, 2020; Subbiah et al, 2020; Drilon et al, 2023; Kacew et al, 2020; Matulewicz, 2020; Rizzo, 2022; Necchi, 2020

TYRA-300 showed greater potency for FGFR3 than other isoforms

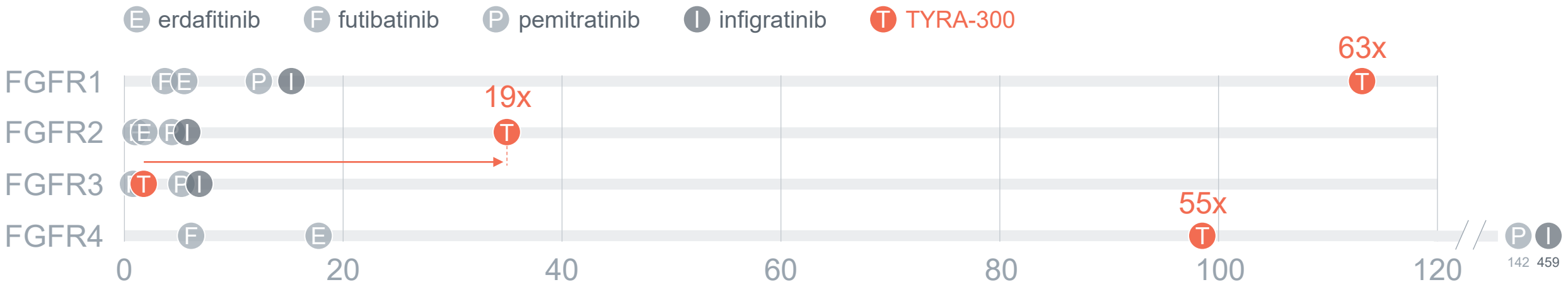


	TYRA-300	FGFR3 selectivity
FGFR3	1.6	1.0x
FLT4	2.1	1.3x
FGFR2	6.5	4.0x
FGFR4	11.0	6.9x
JAK2	35.5	22x
LTK	65.1	41x
FGFR1	108	68x
FLT1	201	126x
JAK3	206	129x

TYRA-300 was profiled in a scanMAXSM (KINOMEscan) screen, IC50 data generated by Reaction Biology Inc.

TYRA-300 was more selective for FGFR3 than pan-FGFR inhibitors

Selectivity observed for TYRA-300 vs. approved or late-stage clinical compounds: *in vitro* Ba/F3 Cellular IC_{50} (nM)

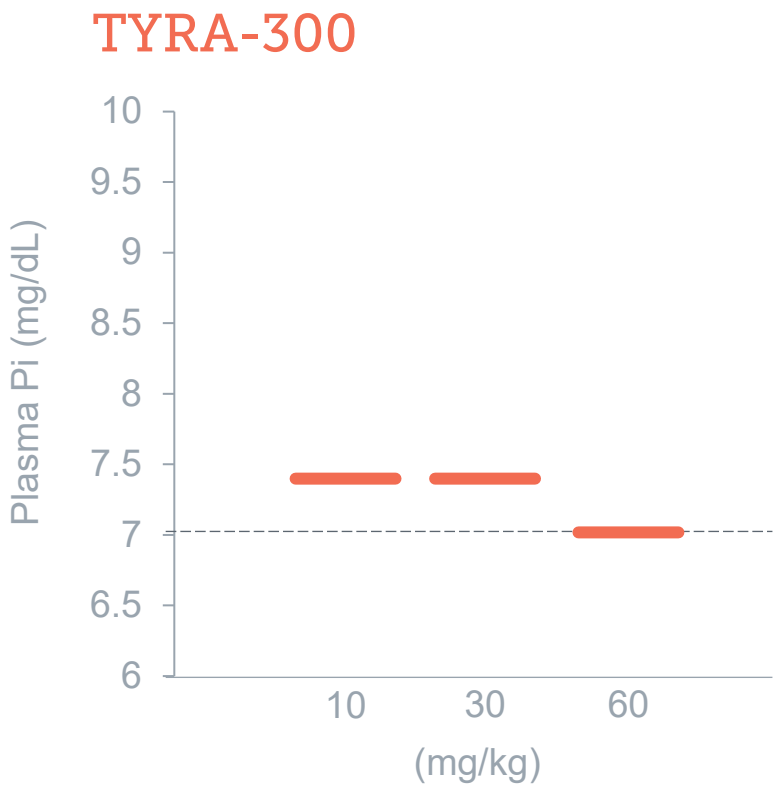


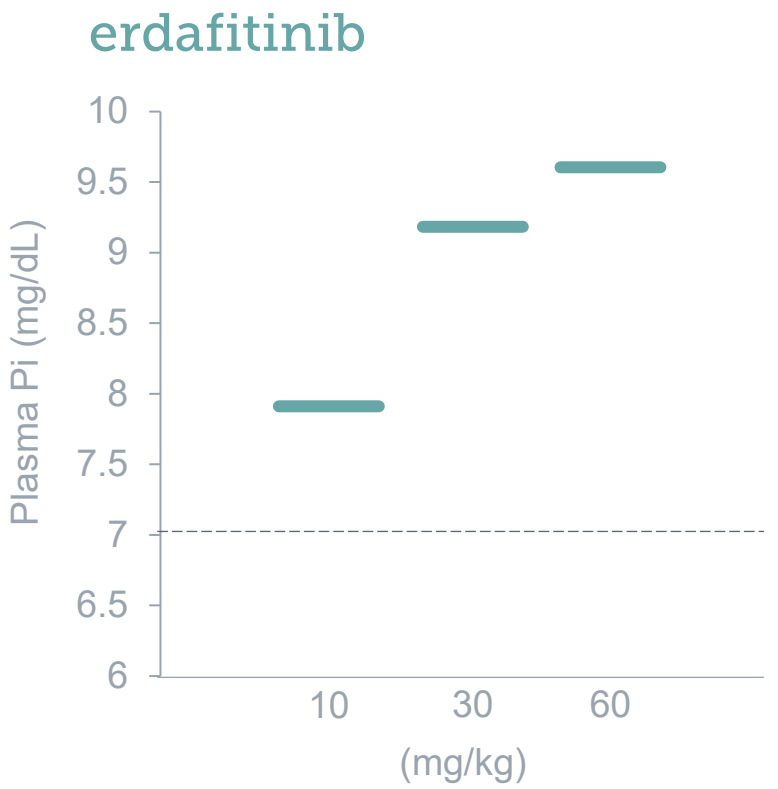
	E	F	P	I
FGFR1	4.2x	4.9x	2.4x	2.2x
FGFR2	1.4x	1.3x	0.8x	0.8x
FGFR4	14x	7.6x	27x	67x

All experiments conducted under identical conditions, tested in duplicate.

TYRA-300 did not elevate phosphate relative to erdafitinib *in vivo*

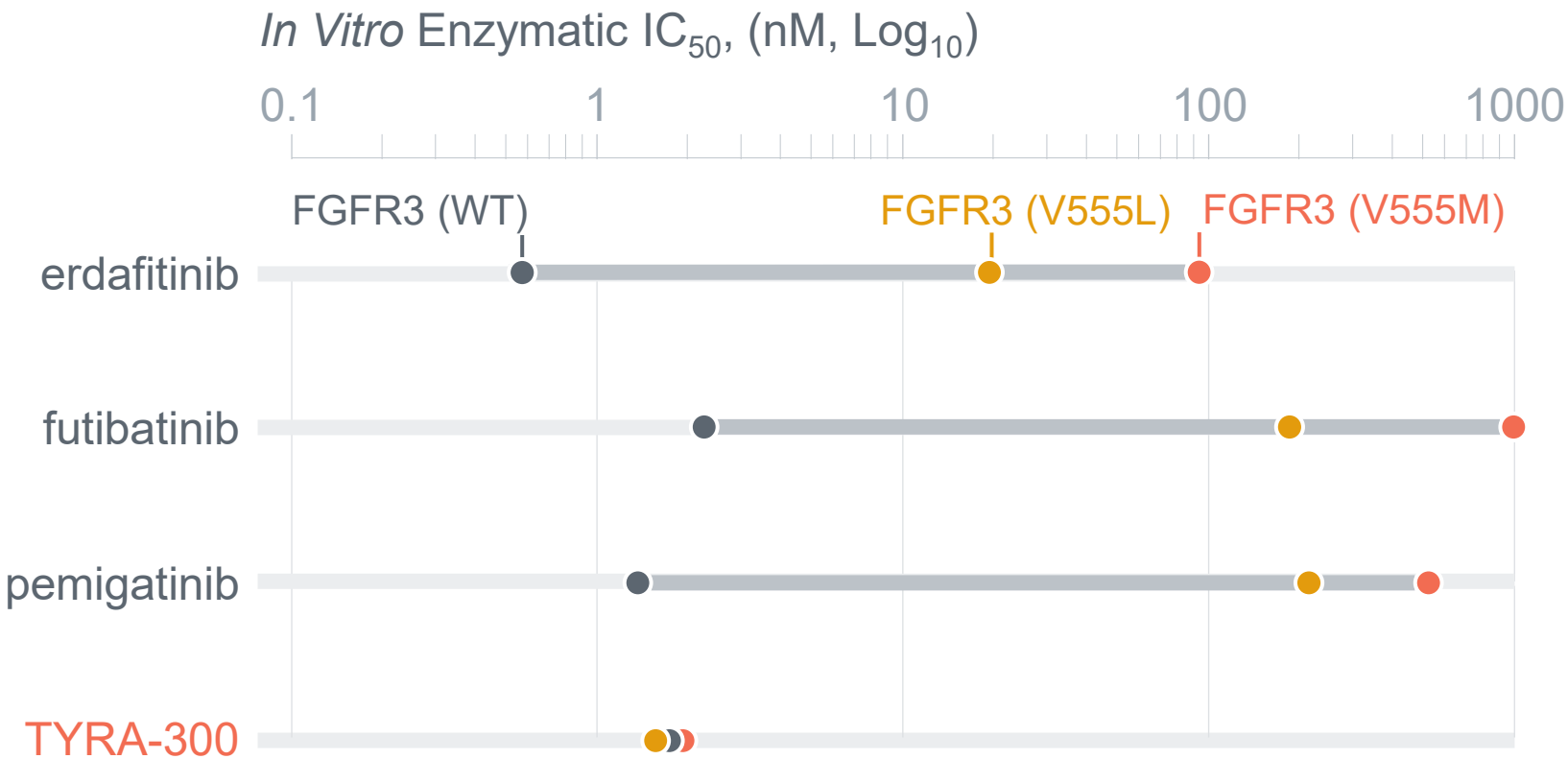
Rat plasma phosphate at 24 hours after single dose[1]





1. N=4 per group, pooled rat plasma; dotted line = pre-dose phosphate value of 3 dose groups

TYRA-300 retained potency against FGFR3 gatekeeper mutations

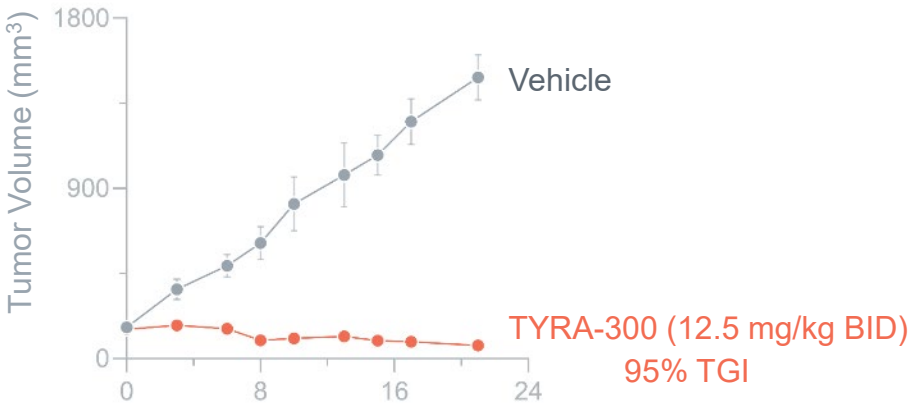


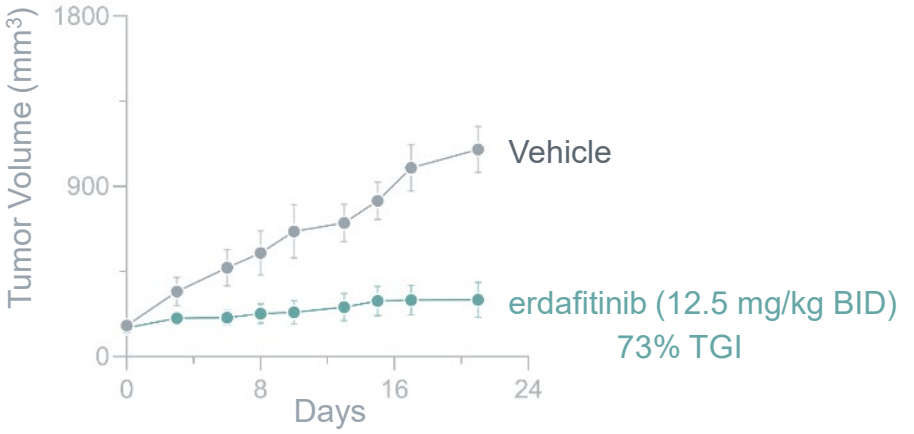
TYRA-300 was designed to avoid the "back pocket" where gatekeeper mutations emerge.

All experiments conducted under identical conditions, same day, tested in duplicate.

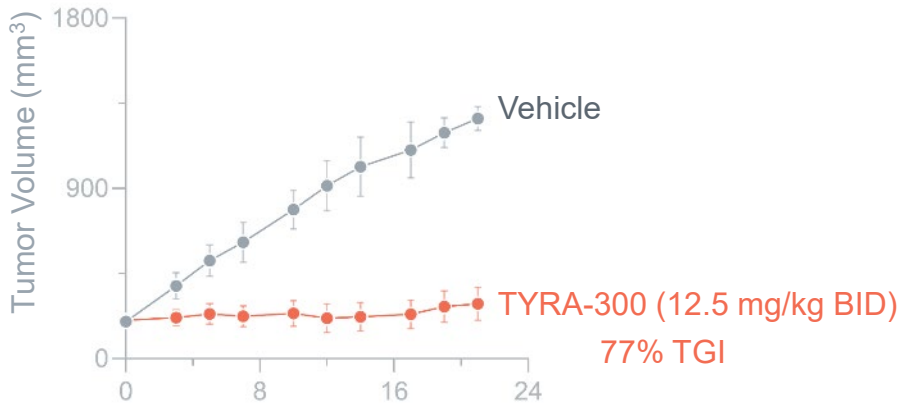
TYRA-300 showed activity *in vivo* in bladder cancer models

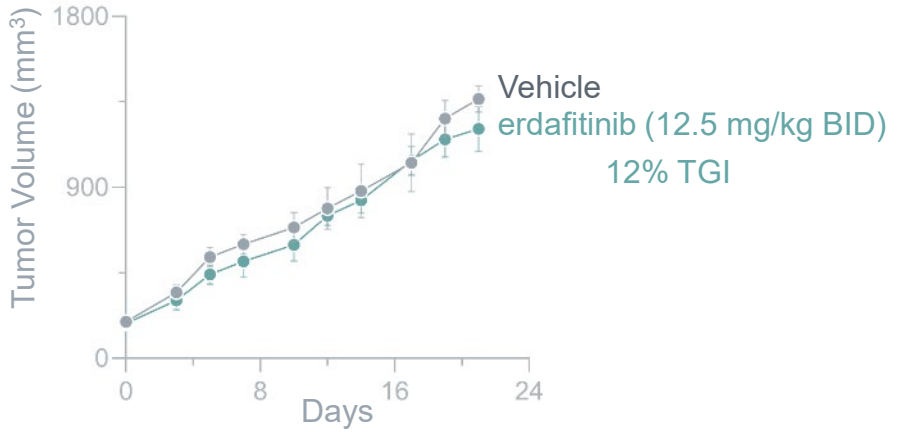
Bladder Cancer Xenograft *FGFR3:TACC3*



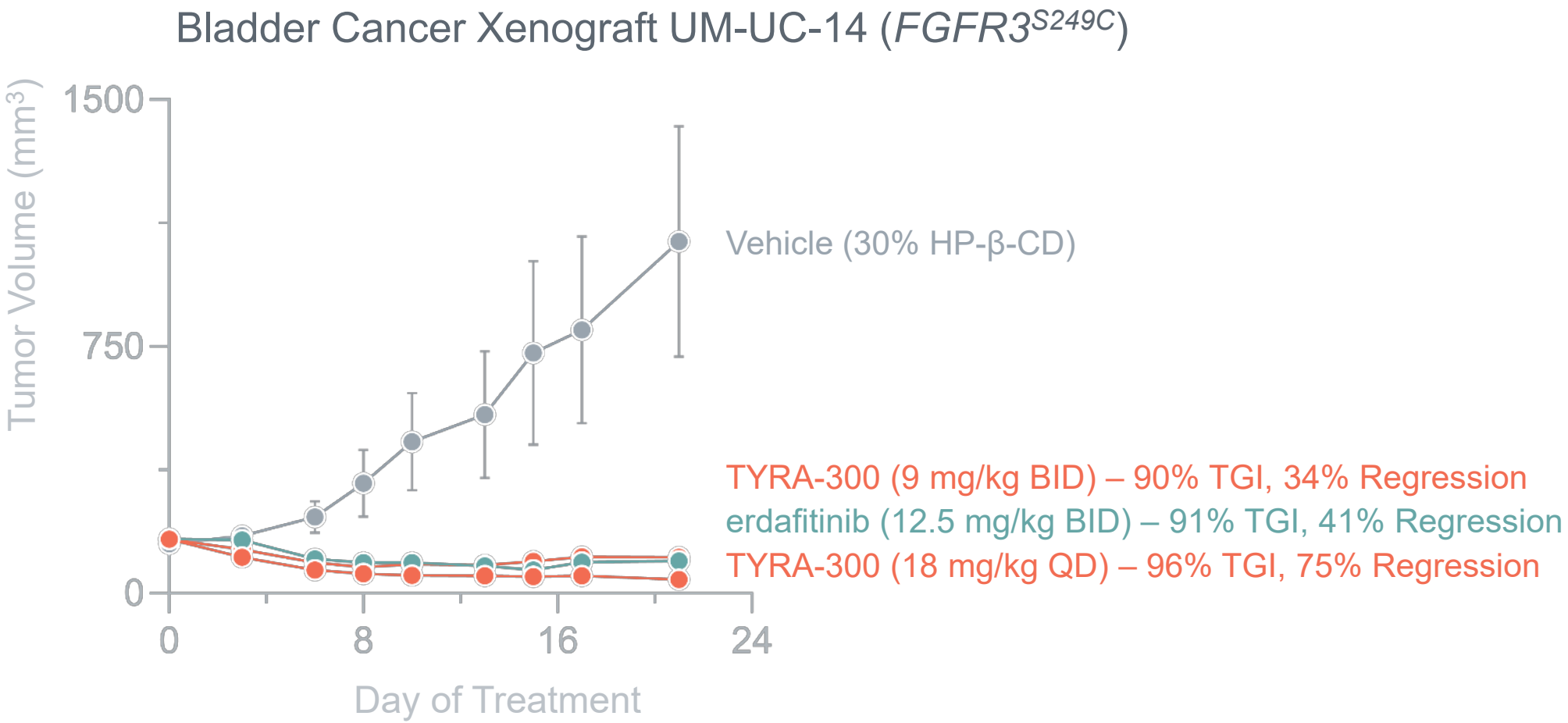


Bladder Cancer Xenograft *FGFR3:TACC3-V555M*





TYRA-300 regressed tumors in key urothelial xenografts

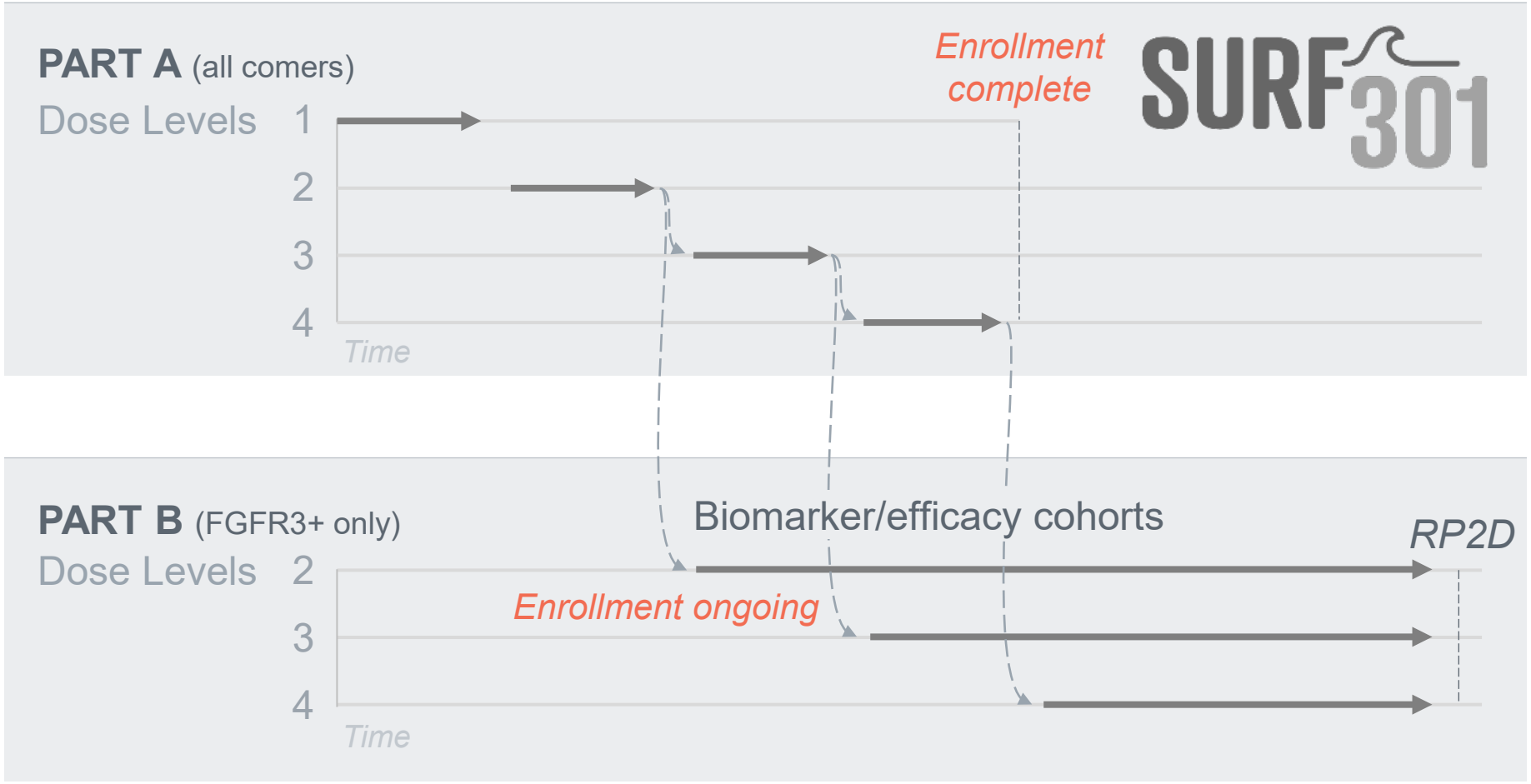


Our Phase 1 trial is designed to identify the RP2D

Illustrative

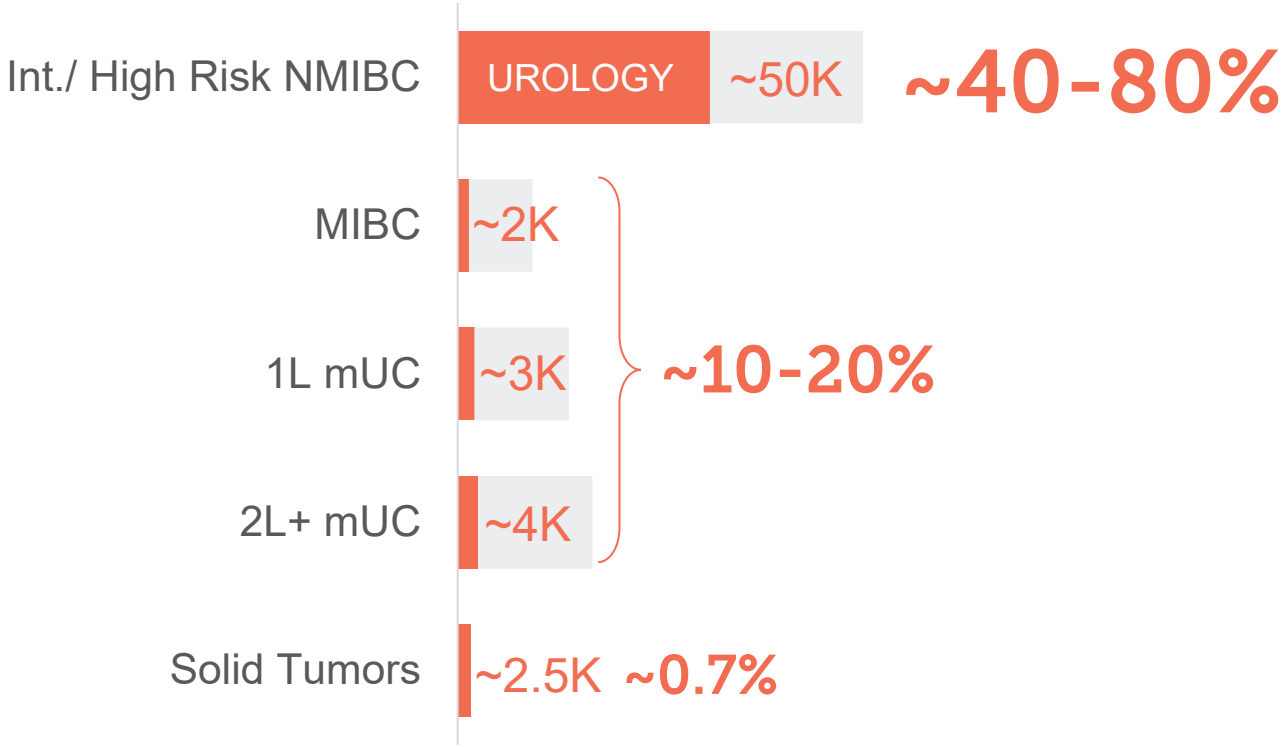
What is the MTD?

What is the RP2D?



The estimated addressable FGFR3 patient population is large

Estimated 2022 US FGFR3+ Addressable Population[1]



DRIVER MUTATIONS

S249C,
R248C,
Y373C,
G370C,
FGFR3-TACC3 fusion

CDx

Liquid Biopsy
NGS
Fusion detection

1. Bladder figures represent potential annual diagnosed incident and recurrent case estimates by addressable disease stage; Solid tumors figure based on annual deaths
Source: Clarivate Analytics; Kacew, 2020; Knowles, 2014; Mayr, 2022; van Rhijn, 2010; Murugesan, 2022

Erdafitinib has demonstrated PoC for FGFR inhibition in UC

	ERDAFITINIB (ORAL)	TAR-210 (PRETZEL)
Intermediate-Risk NMIBC	83% CR[1] (n=18)	90% CR[2] (n=31)
High-Risk NMIBC (Papillary)	77% 12mo RFS[3] (n=49)	90% RFS[2] (n=21)
High-Risk NMIBC (CIS)	73% 6mo CR[4] (n=11)	
1L mUC	55% ORR with PD-1[5] (n=44)	
2L/3L mUC	35.3% ORR, 12.1mo OS[6] (n=136)	
Unmet Need	Durability and Tolerability	Administration

Note: Exposure refers to median exposure to therapy 1. Daneshmand, 2023 (SUO) 2. High risk cohort represents patients with recurrence after BCG; Vilaseca, 2024 (AUA) 3. High risk cohort represents patients with recurrence after BCG; Catto, 2023 (ESMO) 4. Catto, 2023 (SUO) 5. Siefker-Radtke, 2023 (ASCO) 6. Erdafitinib label; data for patients with FGFR3 alterations

TYRA

TYRA-300^{ACH}

TYRA-300^{ONC}

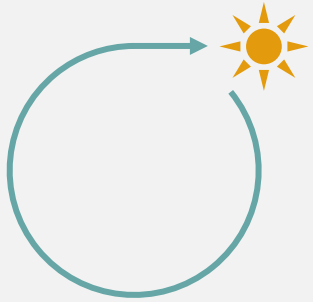
SNÅP

Our rapid approach to discovery continues to generate promising candidates such as TYRA-200

Our unconventional approach accelerates discovery

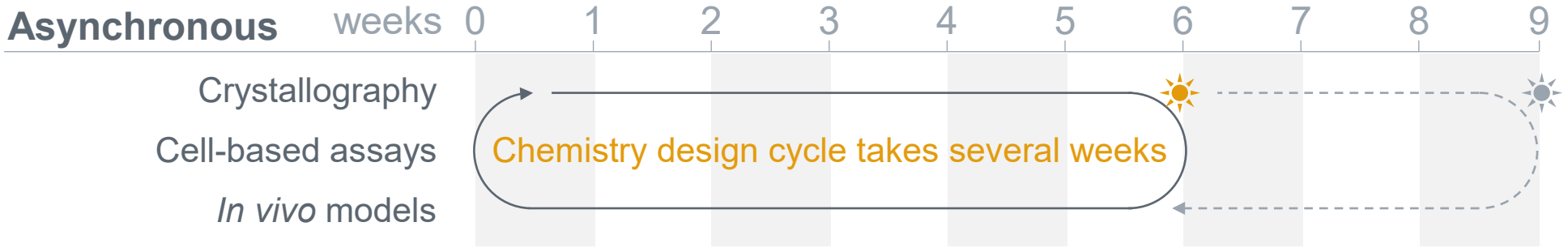
DESIGN CYCLE

Chemistry design

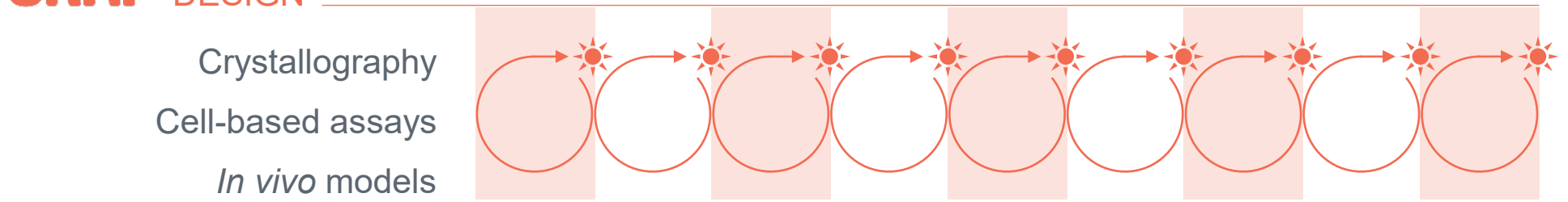


Crystallography
Cell-based assays
In vivo models

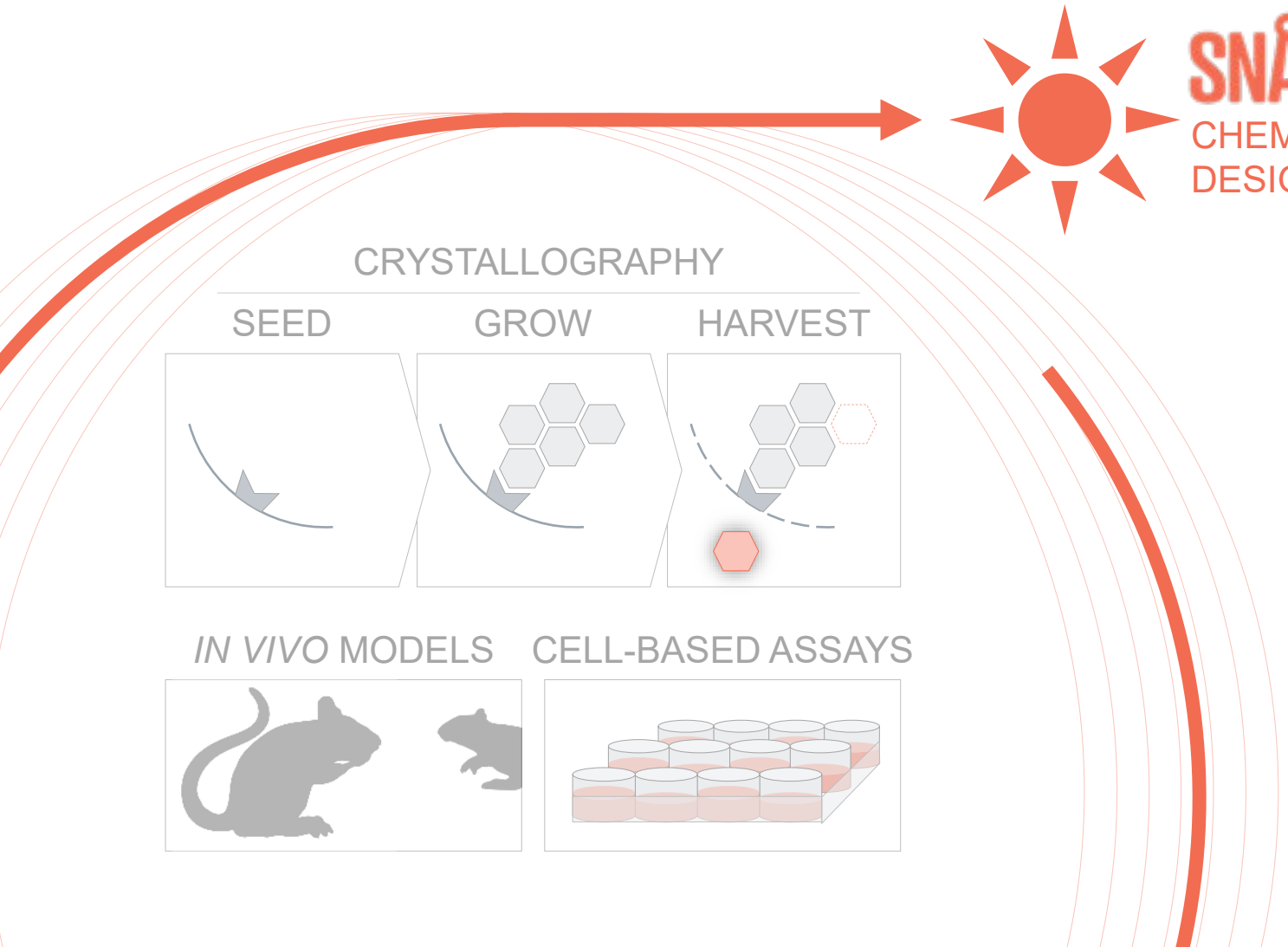
Asynchronous



SNAP CHEMISTRY DESIGN



We’ve optimized the drug design cycle in-house



CRYSTALLOGRAPHY

New compound to structure in as little as 3 days

CELL-BASED ASSAYS

New compound to cellular data in as little as 2 days

IN VIVO MODELS

New compound to initial PD readout in as little as 5 Days

EXAMPLE

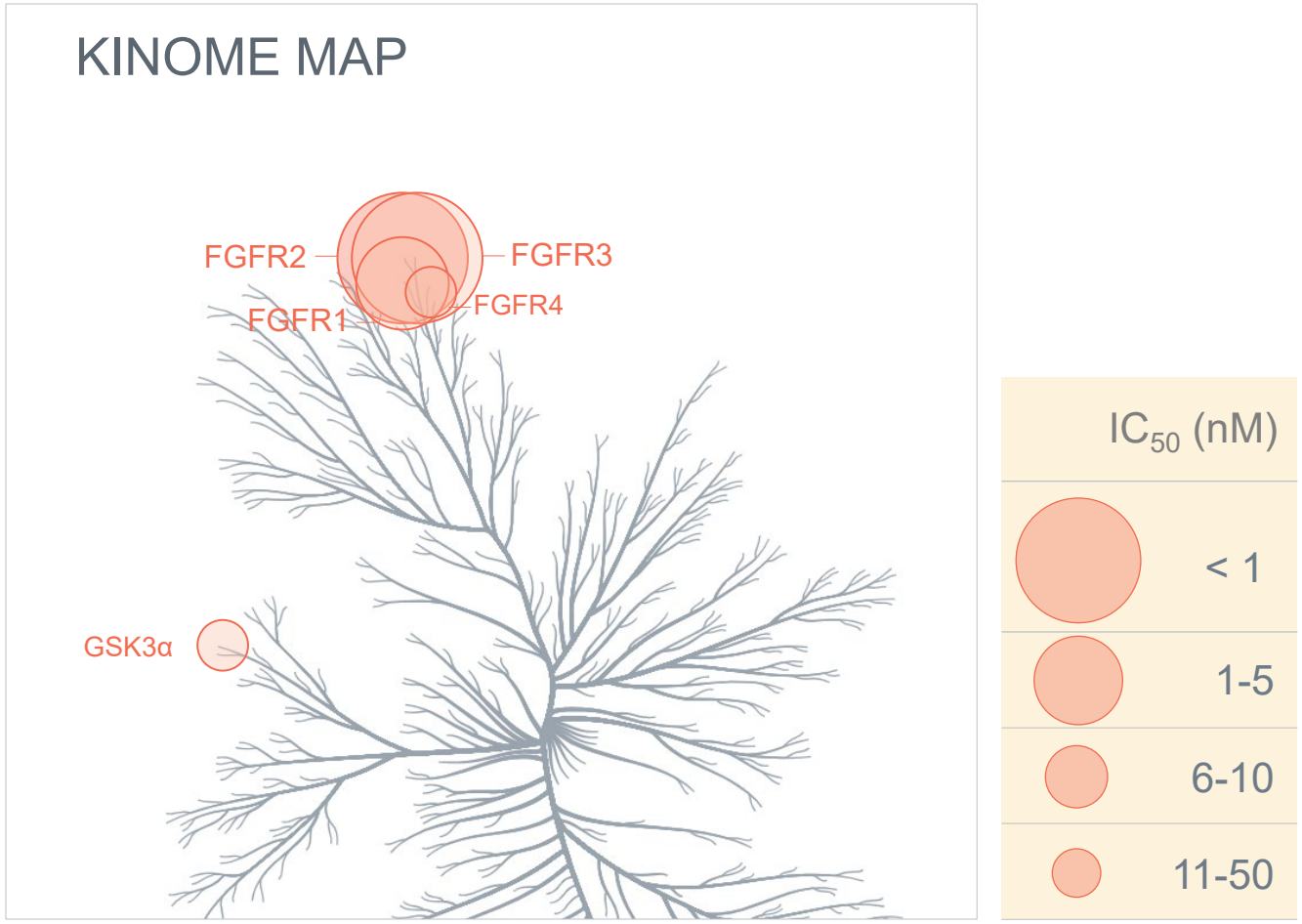
TYRA-200

Intrahepatic cholangiocarcinoma (ICC)

Designed to be gatekeeper- and molecular brake-agnostic and FGFR4-sparing

Potential gateway to additional solid tumor development programs

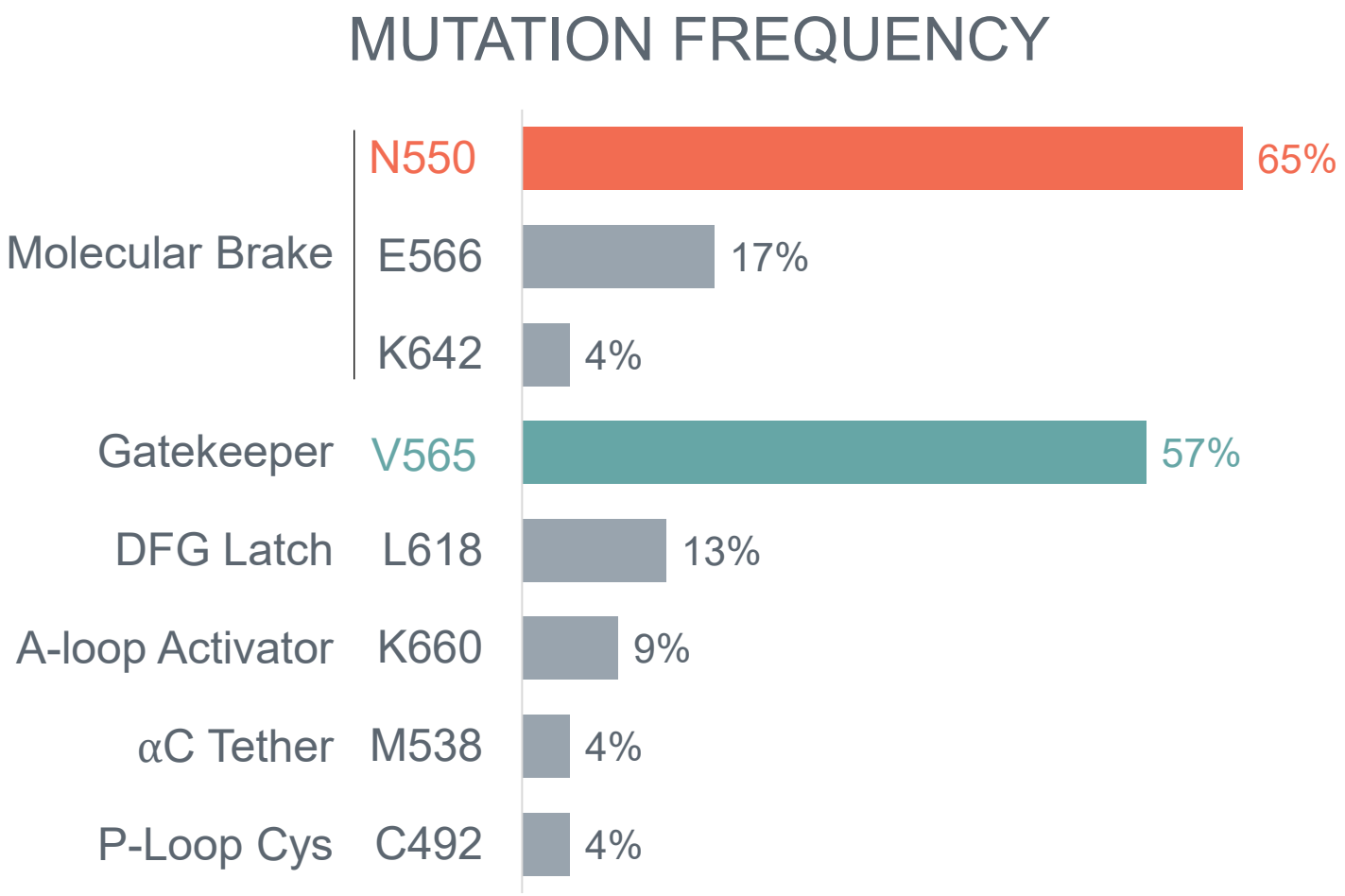
TYRA-200 showed high selectivity for FGFR1/2/3, sparing FGFR4



	TYRA-200	FGFR2 selectivity
FGFR2	0.47	1.0x
FGFR3	0.66	1.4x
FGFR1	1.8	3.8x
FGFR4	30.5	65x
GSK3α	35.6	76x

TYRA-200 was profiled in a scanMAXSM (KINOMEscan) screen, IC50 data generated by Reaction Biology Inc.

Polyclonal acquired drug resistance occurs often in FGFR2



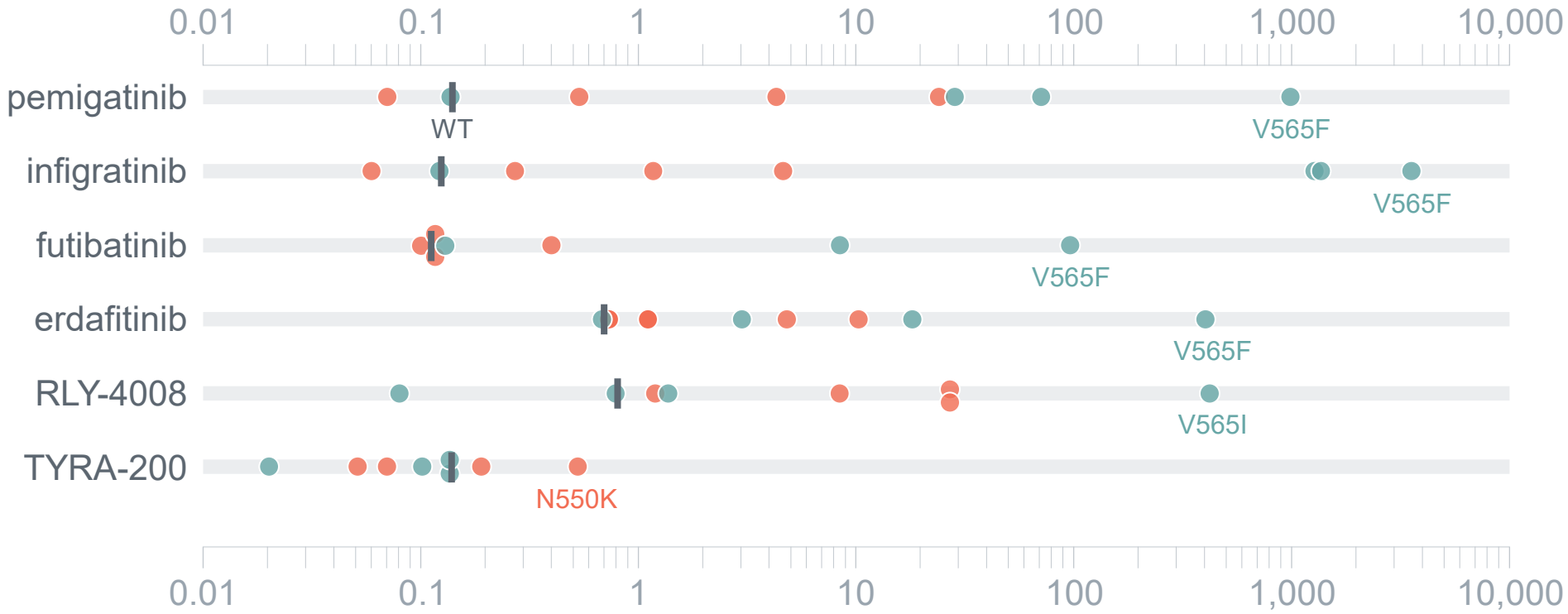
Over 50% of patients who recur on FGFR therapy have gatekeeper and molecular brake mutations

Data presented at EORTC (October 2020); N=46. Evaluable FGFR2-fusion Patients (ICC) on FGFR therapy w/ post-progression biopsy

TYRA-200 retained potency for key acquired resistance mutations

In Vitro Enzymatic IC_{50}, (nM, Log_{10})

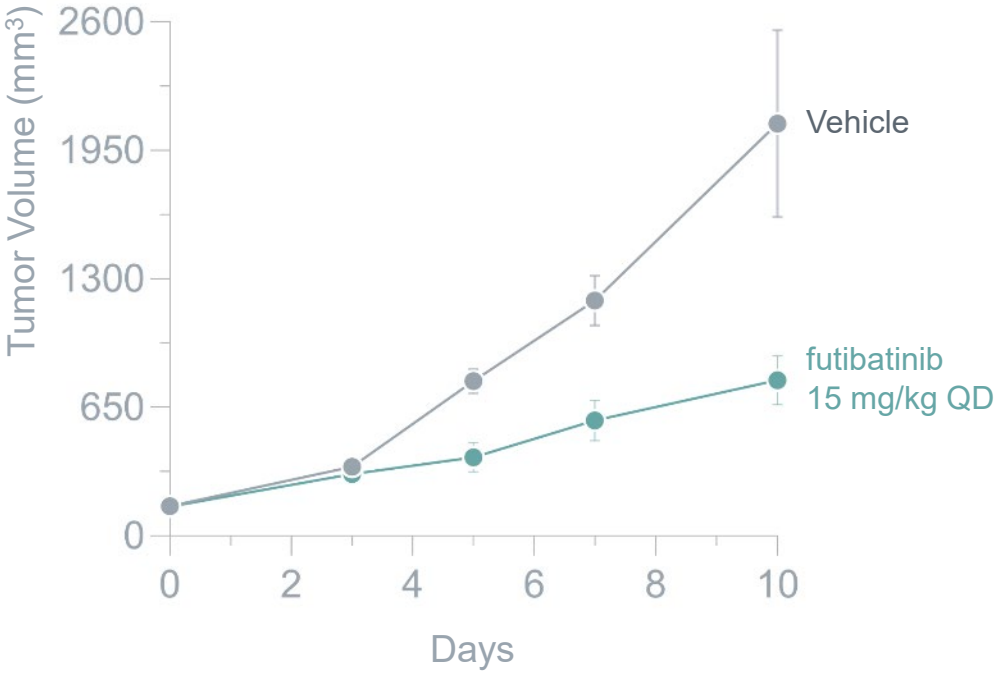
Variants tested: **WT, N550D, N550H, N550K, N550T, V565F, V565L, V565I**



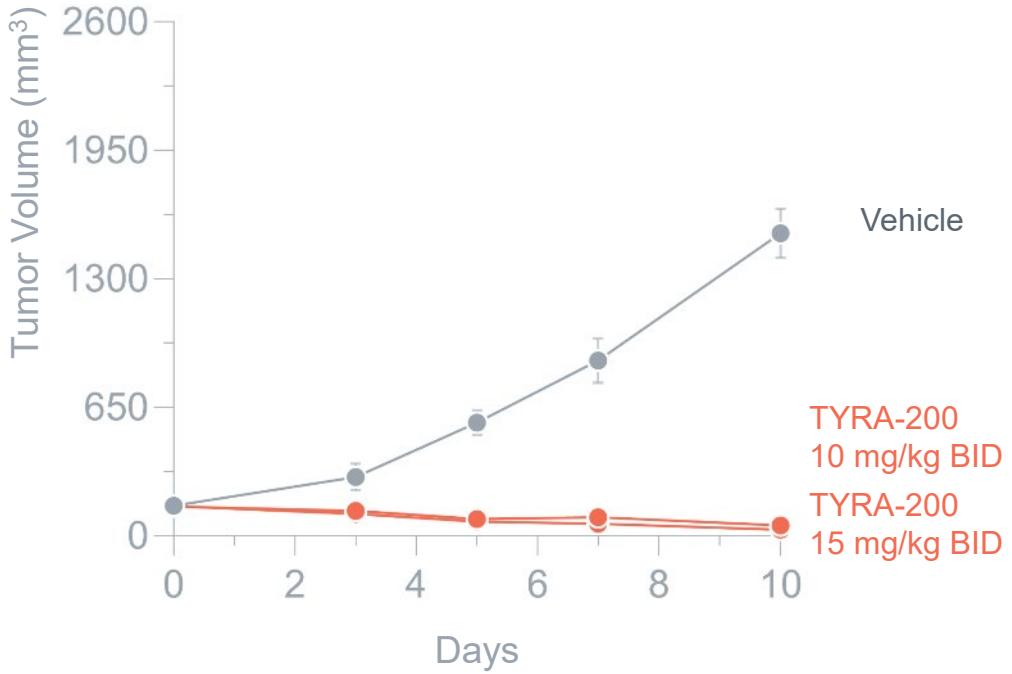
Enzymatic IC50 measurements generated at Reaction Biology Corp using Tyra enzymes.
All experiments conducted under identical conditions, tested in duplicate.

TYRA-200 regressed tumors with gatekeeper mutations

Ba/F3 FGFR2 V565F Allografts

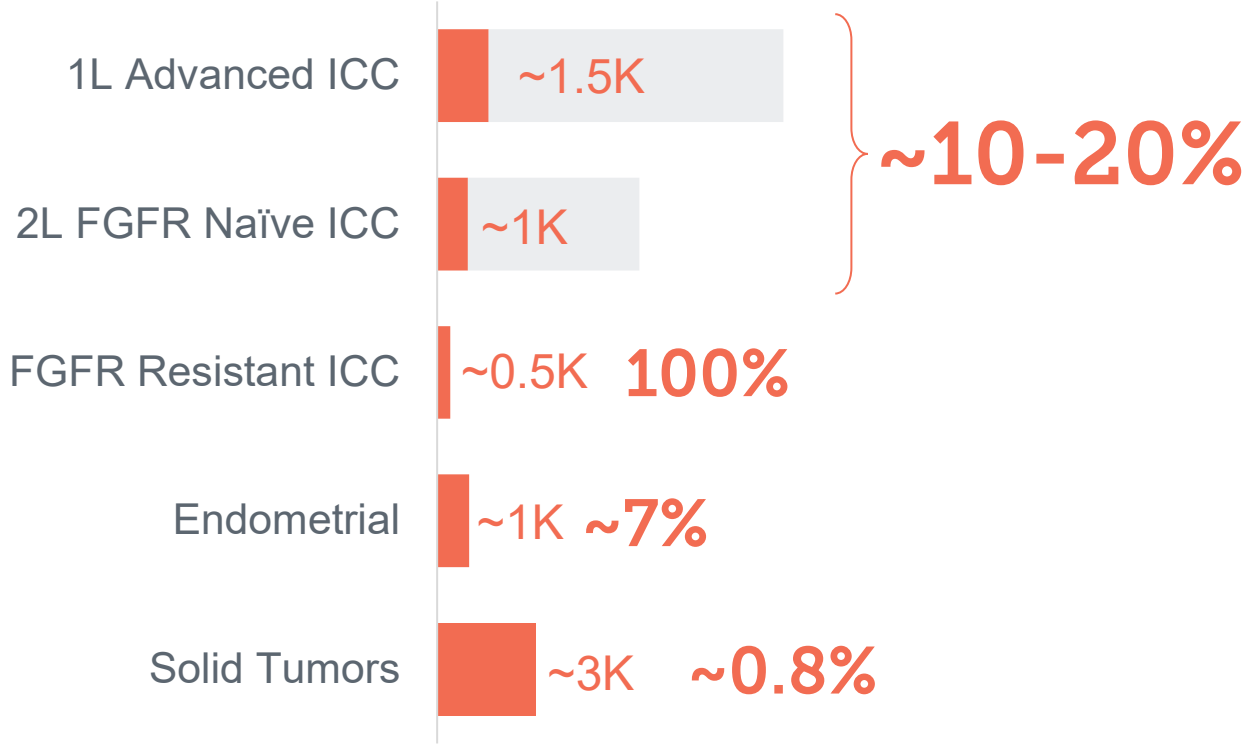


Ba/F3 FGFR2 V565F Allografts



TYRA-200 has multiple opportunities across FGFR2+ solid tumors

Estimated Potential Annual US FGFR2+ Addressable Population[1]



Driver mutations

Rearrangement+,
$N550^{MB}$,
K650E,
S252W,
Y375C,
C382R

1. ICC figures represent potential annual incident and recurrent case estimates by addressable disease stage; Solid tumors figure based on annual deaths
Source: SEER; Cleary, 2021; Conway, 2022; Oh, 2022; Goyal, 2020; Murugesan, 2022; Company Research

Acquired resistance is a key unmet need in FGFR2+ ICC

	ADDRESSABLE (US)[1]	LEAD OPTION	UNMET NEED
1st Line	~1.5K	CPI + Chemo	Only ~27% of patients respond; Increased PFS (Durva+Gem/Cis: 7.2mo[2])
2nd Line	~1K	FGFR2 Inhibitors	Increased PFS (futibatinib: 8.9mo[3]) ~67% of FGFR2i responders relapse with resistance mutations[4]
3rd Line	~0.5K	Chemo or palliative	Polyclonal resistance; need for gatekeeper and molecular brake-agnostic approach

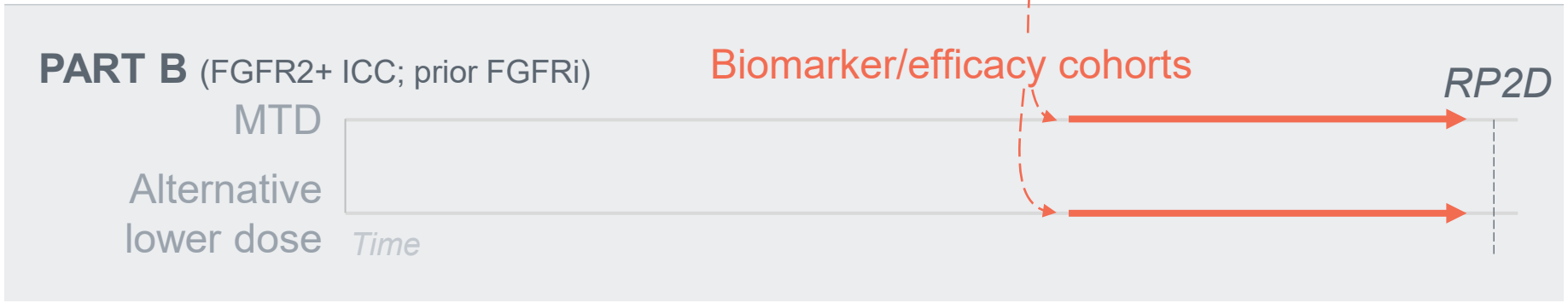
1. Represents estimated potential annual incident and recurrent case estimates by addressable disease stage 2. Oh et al, 2022; 3. Data presented at at ASCO (June 2022); N=103; 4. Data presented at EORTC (October 2020); N=46. Evaluable FGFR2-fusion Patients (ICC) on FGFR therapy w/ post-progression biopsy

Our Phase 1 trial focuses on FGFR resistance patients

Illustrative

What is the MTD?

What is the RP2D?



Our expertise in FGFR biology creates a differentiated pipeline

GENETIC CONDITIONS	Estimated Annual US Addressable[1]	Discovery	IND-Enabling	Phase 1	Phase 2	Phase 3	Anticipated Milestone
FGFR3^{ACH}: TYRA-300	~3K	———	●·····	*Data from FGFR3^{ONC}*	····▶		Submit IND 2H '24
		Potentially leading to additional skeletal dysplasias, including FGFR3-related conditions (HCH, SHOX), and pediatric short stature					
ONCOLOGY							
FGFR3^{ONC}: TYRA-300	~40K	———	———	———	SURF301		Initial Ph1 data in 2H '24
FGFR2^{ONC}: TYRA-200	~5.5K	———	———	———	SURF201		Complete Ph1
FGFR4/3^{ONC}: TYRA-430	~9K	———	●———				Complete IND enabling

TYRA retains an active FGFR3 discovery program.
1. Represents FGFR3/FGFR2/FGF19+ incidence and relapses for TYRA300/200/430, prevalence for ACH

Here's a snapshot of TYRA

Differentiated assets

Precision small molecules targeting large opportunities in FGFR biology

Lead asset: TYRA-300, the first oral FGFR3-selective inhibitor in the clinic

Accelerated design

SNAP CHEMISTRY DESIGN

NASDAQ: TYRA

CASH Mar 31, 2024: $382.5M

UP NEXT

TYRA-300^{ONC}: plan to report initial Ph1 results at a scientific congress in 2H '24

TYRA-300^{ACH}: plan to submit IND in 2H '24

TYRA-430^{ONC}: plan to complete IND-enabling studies

ACH: Achondroplasia; ONC: Oncology
All TYRA small molecule inhibitors are for investigational use only